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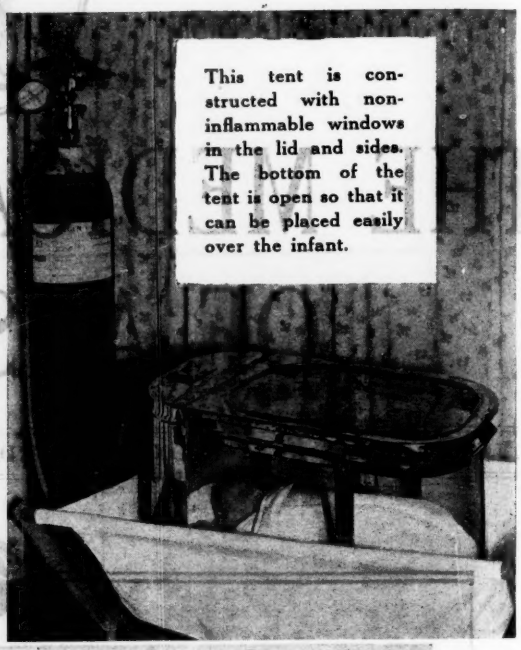
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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—36TH YEAR. SYDNEY, SATURDAY, JANUARY 1, 1949. No. 1.

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THE BACTERIOLOGICAL DIAGNOSIS OF SALMONELLA INFECTIONS.

By I. M. MACKERRAS and M. J. MACKERRAS,

Queensland Institute of Medical Research, Brisbane.

DURING an investigation of Salmonella infections in Brisbane over the past ten months, we have had occasion to examine 1506 specimens of fæces from 784 individuals (mostly infants), as well as a variety of other materials, 223 strains of Salmonella belonging to 13 species being isolated. The staff was small and field laboratory facilities were all that were available, so we had to use simple methods and labour-saving devices in order to get through the work. We received considerable help in these respects from the description by Ewing and Bruner (1947) of their experience with a United States Army laboratory in Italy, though we found it convenient to modify their procedures in some ways. We therefore feel that it may be helpful to other laboratories briefly to record our findings.

This discussion will be confined to the problem of isolating Salmonella organisms from fæces, though the methods are applicable *mutatis mutandis* to other materials. Furthermore, the limit set to the work was to place an organism in its serological group, the culture being then sent to Miss Nancy Atkinson at the Institute of Medical and Veterinary Science, Adelaide, for final identification.

Selection of Materials.

At first cultures were made in the ward within a few minutes of the specimen's being passed; but experience showed that Salmonella could be grown from fæces up to five hours old, so three hours was set as the safe normal limit for receipt in the laboratory. There was usually no difficulty in keeping approximately to this limit.

Infants' fæces were sent to us wrapped in the napkin into which they had been passed, those of older subjects in ice-cream buckets. These can be purchased for a few shillings per hundred, have good lids, and were found by army laboratories during the war to make safe, easily disposable containers. Rectal swabbings were not practicable at the time.

We found no relation in acute cases between the macroscopic appearance of the stool or the presence of cellular exudate and the likelihood of isolating Salmonella organisms from it. We lack statistics, but have the impression that we got more positive results by simply "rubbing up" a general sample of the material than by selecting and washing a flake of cellular mucus. In passing, the commonest exudate in acute Salmonella infections was "indefinite", though a few exudates were "bacillary" by Boyd's classification. These contrasts with bacillary dysentery were striking, but even more striking was the fact, as will be shown later, that it was easier to isolate Salmonella from the formed stool of the convalescent or the carrier than from the fluid fæces of the patient in the acute stage. We suspect that many cases of salmonellosis in infants may be missed by failure to examine the fæces late enough in the disease.

Initial Cultures.

All specimens were plated directly on a selective medium and emulsified in tetrathionate broth. For direct plating we had available "Difco" SS agar, desoxycholate citrate agar and McConkey's agar. All gave good colonies, but the first two took a heavier inoculation without overgrowth than the McConkey medium. Selection of "SS" agar rather than desoxycholate citrate agar as our standard medium was rather a matter of availability than of any discernible difference between them.

The tetrathionate broth was also incubated for twenty-four hours (sometimes forty-eight or seventy-two), and then plated on SS agar. The results of 200 consecutive Salmonella isolations in which both methods were used were as follows: "positive" from both direct plate and tetrathionate broth, 99; "negative" from direct plate, "positive" from tetrathionate broth, 87; "positive" from

TABLE I.

Kligler Medium.	Urea Decomposition.	Lactose Fermentation.	Glucose Fermentation.	Mannite Fermentation.	Sucrose Fermentation.	Indole Production.	Motility.	Gram Staining.
"R/YBG"[1]	–	–	AG[2]	AG[2]	–	–	++	–

[1] Salmonella reaction.

[2] Acid and gas.

direct plate, "negative" from tetrathionate broth, 14. Thus 93% of the fæcal infections found were revealed by the tetrathionate broth and only 57% by the direct "SS" plate, which is in accord with experience elsewhere (Hynes, 1942; Galton and Quan, 1944; Draper, 1945). The direct plate has a compensating advantage in that, when the result is positive, it can be reported twenty-four hours earlier than that obtained from tetrathionate broth.

Preliminary Treatment of Colonies.

Isolated Salmonella colonies on "SS" plates are large (two millimetres or more in diameter) and brownish, with a darker centre. Their identity could usually be suspected, but many colonies of paracolon bacilli and Proteus species were indistinguishable to our eyes. Eight to ten colonies were inoculated in Kligler's agar and incubated for twenty-four hours. This medium was invaluable in giving a quick distinction between cultures which might be Salmonella and those which certainly were not. The "Salmonella reaction (R/YBG)"[1] never failed in our series of true Salmonella organisms, and was duplicated by only a few rare paracolon bacilli and approached by some Proteus organisms.

That the differentiation of Salmonella from Proteus was a matter of degree rather than of kind presented a real difficulty, because members of the latter genus were present in almost every specimen. To overcome it we replaced the glucose of Kligler by mannite and obtained a very good contrast; the Salmonella reaction was "R/YBG",[1] the Proteus reaction was "R/B".[1] The "mannite-Kligler" medium might have many uses, provided one remembered that Shiga or Schmitz dysentery bacilli would give an "R/R" reaction.[1]

A second valuable aid, when ordinary Kliger medium was used, was to test all "R/YBG"[1] cultures on a urea medium as recommended by Ewing and Bruner. We preferred the solid medium of Christensen (1946) to the standard test on fluid medium of Stuart, Van Stratum and Rustigian (1945). All Proteus cultures decomposed the urea within two to four hours, frequently in less than an hour, so cultures tested in the morning could be either discarded as urease-producing Proteus or inoculated into sugars after lunch. The preliminary diagnostic usefulness of this procedure decreased when the "mannite-Kligler" medium was developed; but we retained it as a routine test for our Salmonella cultures, for it immediately revealed contamination with Proteus which occurred occasionally during "fishing" of the colony, and which might not otherwise be detected.

The urea medium was also useful when it was desired to test large numbers of colonies on a crowded plate or from a replate of a suspected contaminated Salmonella colony. Thirty or forty colonies could be picked off quickly onto urea-agar, the results read within two hours, and the "urease-negative" cultures (perhaps only two or three out of the many) tested further without loss of time.

We tried phenol-red-tartrate broth as a means of detecting *Salmonella bovis-morbificans* in overgrown plates, inoculating heavily, replating from tubes that showed acid and gas, and testing resulting non-lactose-fermenting colonies on urea-agar. We gave it up when we found that some strains of this organism were tartrate-negative.

Confirmatory Tests.

Colonies giving an "R/YBG" reaction[1] on "mannite-Kligler" medium (or "R/YBG"[1] on ordinary Kligler medium and a negative response to the urease test) were inoculated into four sugars and peptone water. The reactions given by all our strains of Salmonella (except one which was anaerogenic) are shown in Table I. We found no strain that did not produce abundant sulphuretted hydrogen and none that was mucoid (compare Atkinson *et alii*, 1947). All strains giving these reactions and a strongly positive response to the slide test with Commonwealth Serum Laboratories' polyvalent Salmonella agglutinating serum were subsequently confirmed by Miss Nancy Atkinson as Salmonella strains, whereas none of those which gave the reactions but a negative or equivocal response to the slide agglutination test were subsequently found by her to be Salmonella strains.

Slide tests with pure "O" group agglutinating serum provided supporting evidence as well as epidemiological guidance. However, we soon learned not to rely implicitly on grouping, for other group C species sometimes appeared unexpectedly and disconcertingly in the midst of an epidemic due to *Salmonella bovis-morbificans*.

Our experience of species of Salmonella is still not wide, but it encourages us to think that these limited procedures in identification are reasonably reliable for ordinary laboratory purposes.

Reliability of Methods.

We felt that we were picking up most of the Salmonella infections, but the occurrence of clinically typical cases in which we could not isolate Salmonella, of cases in which repeated examinations were necessary before a positive result was obtained, of one post-mortem examination on a patient with a Salmonella in the fæces on the day of death, but from whom none were isolated from the small or large intestine after death, and other similar experiences, showed that an analysis of reliability was highly necessary. Moreover, there seem to be few such assessments in the literature to guide one in the standards one should expect.

We had the strong impression that it was more difficult to diagnose salmonellosis than to follow the infection through convalescence. This was confirmed by analysis. In acute infections with *Salmonella bovis-morbificans*, diagnosis was made as shown as follows:

At first examination	84 cases (68%)
At second examination	21 cases (17%)
At third examination	3 cases (3%)
At fourth examination	10 cases (3%)
At fifth examination	1 case (1%)
At sixth examination	3 cases (3%)

Thus, 198 examinations were required to diagnose 122 cases, a reliability of 61%.[1]

In contrast, 59 of these patients were followed until three consecutive attempts at isolation were unsuccessful. We thus obtained individual series of positive results, sometimes interspersed with negative results, before the final three negative results eventuated. Including all attempts at isolation from the one succeeding the first positive result to the last positive result, we have 235 positive results and 49 negative results, a reliability of 83%. The difference between the early (61%) and late (83%) figures is highly significant ($P < 0.001$).

The character of the stool naturally changes as the infection progresses, the trend from fluid to solid being irregular, chiefly due to recrudescences. We therefore

[1] R = red slope; Y = yellow butt; B = much blackening; G = gas bubbles.

[1] Our material is not sufficiently representative to analyse diagnostic reliability in relation to time from onset or to other species of Salmonella.

analysed character without reference to stage, and obtained an almost equally significant association (Table II):

TABLE II.

Type of Stool.	Positive.	Negative.
Fluid or semifluid[1] ..	108 (66%)	56 (34%)
Solid or pasty[1]	184 (80%)	46 (20%)

[1] $P=0.001$ for difference between "fluid" and "solid" groups.

Consequently, we cannot say whether the character of the stool or the stage of the disease was responsible for our findings.

All these figures are subject to qualification. In the first place, they are based on the assumption that infected persons excrete Salmonella organisms continuously. This may not be true; but we feel it unsound to assume the contrary.

Secondly, the greater difficulty in diagnosis as compared with subsequent follow-up may have been due to the fact that some patients had originally had an enteritis from another cause and had acquired a Salmonella infection while under treatment. There is evidence that a considerable number of patients acquired a Salmonella infection in hospital, and, in the specific field of enteritis, four patients with *Salmonella typhi-murium* infection, two with *Salmonella adelaide* infection and one with Flexner dysentery subsequently showed *Salmonella bovis-morbificans* in the stools, one with a *Salmonella bovis-morbificans* infection developed a *Salmonella adelaide* infection, and one with *Salmonella adelaide* infection developed a *Salmonella typhi-murium* infection. For this reason we have excluded from the earlier tabulation one child with enteritis, from whom twelve negative results to attempts at isolation were obtained by the hospital laboratory and three by ourselves during the month before *Salmonella bovis-morbificans* was isolated from his fæces. However, we do not feel justified in excluding any of those with five or fewer negative responses in the absence of a demonstrable other cause for their primary condition, though we may suspect that they may not all have been suffering from primary salmonellosis. Studies now being developed may throw further light on this question.

There is another consideration, which inclines us to the view that much of the unreliability in early examinations was real. In the plates (whether direct or from tetrathionate broth) in acute cases there was usually a great variety of non-lactose-fermenting organisms (Proteus, paracolon bacilli of many kinds and sometimes Pseudomonas abounded—even coliform bacilli were often numerous) and Salmonella colonies were often few and difficult to detect; the media did not seem to be selective enough against these types. As the disease progressed towards chronicity or convalescence, the intruders became fewer and the Salmonella organisms more numerous. We repeatedly obtained abundant pure cultures, often for many weeks from the fæces of our recovered carriers. The change was so great as to give the illusive impression that Salmonella had displaced all other organisms from the intestine. We feel that these observations explain a good deal of the difference between early and late figures. We might have done better if we had used a medium containing brilliant green for plating from the tetrathionate broth (Glass and Wright, 1937; Knox *et alii*, 1942; Galton and Quan, 1944); but none of the authors mentioned indicates the type of stool examined.

Clearance Tests.

A word may be said in conclusion about clearance tests. As the ill and recovered children provided the overwhelming preponderance of infected material in the community, it was felt unwise to discharge them from hospital until they had been cleared of infection. We gave the advice, rather arbitrarily, that three consecutive negative results was a workable standard to adopt. In the 59 cases mentioned earlier, there were 10 instances of two consecutive negative results followed by a positive result. Only 21 examinations of 15 patients were made after the third consecutive negative result; none gave a positive result.

Summary.

The procedure used to isolate Salmonella from fæces was as follows: (i) Plate the material directly on "Difco" SS agar and inoculate tetrathionate broth. (ii) Pick off colonies onto mannite-Kligler medium (alternatively, inoculate ordinary Kligler medium and test any suspicious growth for urease production). (iii) Test cultures giving a "Salmonella reaction" for fermentation of lactose, glucose, mannite, sucrose and urea (if not used previously), for indole production, for motility, for reaction to Gram stain, and with polyvalent Salmonella agglutinating serum.

These procedures economized time and materials and gave a reliability of about 60% in acute stages and 80% in later stages of Salmonella gastro-enteritis in infants.

In our experience, diagnostic cultures should be continued to not less than the sixth, and clearance cultures during convalescence to not less than the third, before a Salmonella infection is excluded.

Positive results may be reported in three days from the direct plate in more than half the cases, in four days from the tetrathionate broth in the remainder. An indication may be given twenty-four hours earlier by direct agglutination from either Kligler medium.

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We are especially grateful to Mr. G. Simmons, of the Queensland Department of Agriculture and Stock Animal Health Station, Yeerongpilly, for supplies of group agglutinating sera, and to Miss Nancy Atkinson, of the Institute of Medical and Veterinary Science, Adelaide, for additional agglutinating sera, as well as for the laborious work of identifying all the strains sent to her.

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PAIN AND THE SURGEON.[1]

By A. E. COATES,

Melbourne.

I INVITE your attention to the phenomenon of pain, the primitive symptom, for which the doctor is usually called, and the alarm signal of disease, the surgical removal of which will bring its cessation. I wish to devote most attention in this lecture, however, to a discussion of the treatment of the symptom of pain in certain cases in which its cause cannot be removed. A small part only of such an enormous field can be covered in one lecture.

[1] Read on March 4, 1948, as part of the scientific session of the Royal Melbourne Hospital centenary celebrations.

I would take you to the consulting room or the bedside, there quietly to listen to the patient's complaint. As the history is unfolded, it should be possible to obtain a fairly clear idea of the mode of onset and the progression of symptoms which the patient describes. It may be necessary to guide the patient a little by appropriate remarks, but let us not merely jot down the answers to leading questions. In that way we may be fooled—we may commence with a preconception and unconsciously build up a false diagnosis or, what is more likely, end in utter confusion, send the unfortunate off to this and that department for useless investigation, wasting money and perhaps life-saving time.

A thorough physical examination will be the appropriate prelude to further scientific and laboratory inquiry, and perhaps the latter may not be necessary or, if it is, it will be considered only in the light of the history and examination. Perhaps the doctor is called to see a patient who is in abdominal pain. It may be intermittent. On the arrival of the doctor the pain has gone. What is to do? Wait, stand by like the obstetrician, place the hand on the abdomen, watch the patient's face, feel the hernial orifices and the rectum. Ah! There's the pain coming on again; the examining hand feels the cramping intestine. Timely operation saves the life. But if on a cursory examination nothing much is found—no distension, no fæcal vomiting, no absolute constipation—the doctor may hurry away, leaving instruction for a sedative (a personal soporific) or an enema, only to find on his next visit that the patient is past medical aid.

There is no more deceptive organ than the small intestine; and how intolerant is that viscus of even partial obstruction of its lumen! The rise in pressure straightens out the coils like a garden hose on the lawn, and soon there are added to the original block a number of angular kinks. The venous return is obstructed; œdema and paralysis supervene. Despite advances such as intubation, correction of electrolyte balance and other special methods, it is still true to say that in many cases of small bowel obstruction the longer such a patient lives before operation, the sooner he dies afterwards.

Is there a surgeon here who has not lamented that he did not open the abdomen the day before? Yet we say there were no signs—only the pain. Did an enema relieve the pain? Could the patient pass flatus or have a desire to and be unable? How is the pain? Persistent or recurring colicky central abdominal pain, unrelieved by enema, requires laparotomy. The pulse rate is deceptive. Certainly the classical triad is diagnostic, but to wait for their appearance is to condemn the patient to death. An X-ray examination may show the distended coils when they cannot be felt. A little distension in the left iliac fossa, a local tender spot (obstructed coil or imprisoned appendix) may be suggestive; but the important fact is the pain. When the pain passes away, ileus supervenes, and then the surgeon is often more distressed than the patient. Regurgitated bowel contents, a silent abdomen, a rising pulse rate, general deterioration, and finally peripheral vascular failure are the next phase. At this time may be found a host of interesting chemical changes, a legion of helpers is in attendance, but all to no effect. The time for action is past.

Not always is the pain due to a coil of small bowel tied in a knot, constricted by a band or incarcerated in some obscure part of the abdominal cavity. The vermiform process may occupy a position in the central portion of the abdomen, high up under the mesentery. Especially is this a troublesome problem in the middle-aged and elderly. The original colicky pain is replaced by a deep-seated, dull, constant ache, perhaps in the back. No rigidity is detected, but only a general tumidity. But a well-defined, though deep, tender spot may be located not at McBurney's point, but nearer the mid-line. There may be a little fever or some rise in pulse rate. The bowels may have been lax in the early stages, but are not now open, and an enema is unproductive. The pain persists. Open the abdomen and there, plastered on the under-surface of the mesentery, is a distended appendix, ready to flood the peritoneal cavity with its foul contents.

Another example of persisting abdominal pain is that associated with disease of the biliary tract. The usual bout of colic has passed; but there is a pain, perhaps local under the right costal margin, perhaps central and radiating to the left. The former indicates that the gall-bladder is distended and inflamed, the latter that the pancreas is under suspicion. Persistence of such pain for twenty-four to thirty-six hours, especially if accompanied by a rising pulse rate, abdominal distension, jaundice, shivering, cyanosis, fever or not, usually calls for something more than expectant treatment. It is often said that in all these cases the condition settles down. I admit that it does; but in some unfortunately it becomes permanently quiescent. A distended liver is no longer capable of functioning properly, and decompression of that organ is sometimes an urgent necessity.

The peptic ulcer produces a syndrome of its own. How the picture changes when invasion of the pancreas occurs or obstruction of the pylorus supervenes! The lesser sac may be involved and present little visible evidence—only the gnawing backache and perhaps some pain referred to the left iliac fossa. The collection in the right subphrenic spaces and occasionally the œdema around the common duct may refer pain to the shoulder tip, as may also an amœbic abscess or a torn spleen.

The inflamed sigmoid or the appendix lying on the bladder, may produce symptoms referred to the urinary tract. Viscera may be cut and burnt, but not distended, without the production of pain.

The pain of intestinal colic, though poorly localized, is none the less real. When mesenteries or parietal peritoneum are involved, then is the localization more accurate.

Localization is a function of somatic nerves, and with it appear rigidity and hyperæsthesia. Nevertheless it is my belief that localization in the viscera as a subjective phenomenon is far more accurate than we generally admit. The inflamed appendix is tender whether it is palpated in the mid-line of the abdomen or *per rectum*. A careful interrogation of the patient will usually elicit that the pain is in the vicinity of the diseased organ and not feet away.

The combination of pain and tenderness will indicate the most suitable site for the incision of approach. It is important, however, to determine whether pain is somatic, visceral or referred. Some of us have seen the patient with an old tuberculous condition of the spine and multiple abdominal incisions; happily the tabetic is less frequently seen nowadays to add to our diagnostic difficulties. The angina patient with abdominal symptoms, as also the patient with early basal pneumonia, must be remembered. Contrast the large painless ulcer of the rectum with the exquisitely painful tiny fissure of the anus. One is visceral, the other somatic. A large growth may involve the fundus of the bladder and produce no pain; but let the trigone be involved and then the patient is constantly aware of his trouble. The somatic element here not only localizes the pain, but refers it to superficial parts—penis and perineum.

It is to be noted that the sacral nerves are the only sensory nerves of importance in the bladder. Cutting sympathetic nerves does not cure bladder pain. I subscribe to Morley's views on the general theory of abdominal pain, but the subject is far from being completely elucidated.

With the background of the hollow viscus and its contractions—its cramping contortions and the colic of bowel, bile duct and ureter, uterus and bladder—let us consider the vascular system. The heart and arteries are capable of registering pain of terrible severity. I hesitate to approach this problem, as it is not properly within the scope of surgery and, further, it is the subject of an address by Dr. William Evans. Suffice it to say that an attack on the left stellate ganglion, either by alcohol injection or by excision, has, in my experience, relieved the patient of anginal pain. A medical colleague referred to me in 1938 received injections three times during four years and painlessly carried on a busy practice until the final breakdown. A man, aged seventy-five years, suffering

from intermittent right hemiplegia and precordial pain of effort, has been for the past two years, since stellate ganglionectomy was performed, free from pain and paresis. My experience is limited to six such patients, all referred by consulting physicians.

The posterior approach for injection and the anterior approach for resection have been used. The injection is relatively simple, the resection not so easy; but it is certainly worth while if cerebral symptoms are present. The larger arteries have direct contact with the central nervous system. Medullated nerve fibres probably convey centrally the protest of a great vessel when it is suddenly blocked by an embolus or thrombus. Again, like the viscera, the pain is vaguely localized but none the less insistent. A terrific agonizing pain in the leg is a signal to look to the circulation in the part. Even the smaller arteries record their incompetence — for example, in Buerger's disease. At first occurs claudication pain and later the unrelieved nocturnal pain at rest. With a sudden popliteal block there is the cold, blue, painful leg—icy cold for the lower two-thirds of the leg and foot. In Buerger's disease, in which most of the collaterals have been working overtime, rubor occurs on dependency of the limb, perhaps ulceration of the toes and gangrene. A common error is to consider the condition due to venous and not arterial obstruction. But venous block may produce a tender calf and some œdema, never the unbearable pain of arterial obstruction. It is hardly necessary for us to be reminded that when a patient complains of pain in the legs or feet, not only the reflexes, but the stance, muscle power and sensation should be examined and the pulse in the ankle region should be particularly noted. Local surgical treatment of the ingrowing toe-nail or the ulcer should be eschewed; nothing but misery for the patient and anxiety for the surgeon will follow. A tender spot may be found at the site of arterial block. More commonly there is nothing to indicate the proximal end of the clot.

Not always is the artery alone involved—the veins are sometimes thrombosed as well as the arteries. The mode of surgical attack differs according to the severity and acuteness of the condition. Removal of clot and administration of heparin and dicoumarol are the ideal procedure, but rarely possible because of vascular disease. The sudden block of the popliteal or femoral artery calls for early operation. The vascular tube requires urgent relief, just as the small intestine. If embolectomy is not possible, then resection of a segment of the thrombosed artery and vein must be performed. This may be followed by a lumbar sympathectomy or ganglionic injection. In my experience sympathectomy alone will not save such a limb. Some years ago I had the opportunity to experiment on a patient who sustained a bilateral block of the common femoral arteries. On one side I performed a resection of the blocked artery and vein and then a sympathectomy; on the other side I performed a lumbar sympathectomy only. I was forced to amputate the leg with the unresected artery a few days later.

Not always is a sympathectomy necessary. Two elderly patients last year were subjected to resection of the empty femoral artery below a block. Both did well with regard to pain. One was a diabetic; she suffered mainly from pain and an ulcer of the toe. She is now well. The other was suffering from Ruerger's disease, and I amputated the leg twenty months later. I had performed a sympathectomy on him twelve years earlier. If the leg is too far gone, amputation above the knee may be necessary at once. Here, as in obstruction of the small bowel, time is the factor of importance. It is not necessary to resect all the thrombosed artery; the removal of a segment of it appears to allow proximal collateral vessels to be released from vasoconstrictor influence. How far is the pain itself a warning, how much a vasoconstricting factor? I am convinced that an attack on the main artery of the limb should not be neglected in a doubtful case. The artery should be explored and, if thrombosed, then resected if more conservative measures are not possible. In the slowly developing Buerger's disease, exercise, prohibition of tobacco and the evening noggin of spirits may keep things under control. Lumbar sympathectomy is useful if the disease progresses and no ulcers are present on the toes. As the operation is relatively simple, I dispense with preliminary tests such as the use of fever, hot-box, spinal anæsthesia, thermopile readings *et cetera*. If these tests gave positive results then one would perform a sympathectomy; but if the results were not satisfactory, should sympathectomy be denied? Blocking of the tibial or peroneal nerve with "Novocain" will give useful information. A rise in temperature is measured with a clinical thermometer between the toes.

I have been impressed with the relief of pain following sympathectomy, even in elderly patients suffering from arteriosclerotic ischæmia of the foot. In one patient of middle age, operated upon over ten years ago, walking for a hundred yards produced severe pain. Sympathectomy was followed by a return of potentia and ability to walk four miles to and from his work each day. The disease tends to burn itself out in some cases and it is worth while supporting the circulation during the active phase. Vascularization of the thrombosed vessels may take place. What of the painful ulcerated foot? No rest is possible, dressings are painful and the patient's life is a misery. The foot should be enclosed in ice. This gives great comfort and conserves the part. Section of the posterior tibial, superficial and deep peroneal and sural nerves above the ankle joint provides relief. This can be performed under local anæsthesia and the relief from pain is most appreciated. Incidentally, vasoconstrictors are thus removed from the digital arteries and an increased circulation promotes healing. I have done this for old people in the eighties, for patients with arteriosclerotic ischæmic ulcers, for diabetics, as well as for patients with Buerger's disease. The residual disability in the foot from small muscle paralysis is negligible. The absence of severe trophic effects—perforating ulcers and such like—following distal resection of nerves is in striking contrast to the occurrence of these complications when somatic sensory nerves are damaged or cut proximally, at or about the root ganglion. It sems that the more distally the nerve is cut, the fewer the trophic changes.

One patient, a male, aged forty years, on whom I thus operated sixteen years ago, has now a return of some sensation in the foot, but the other leg had to be amputated below the knee for gangrene thirteen years ago. He walks as well as I do. The disease is now burnt out.

It should be noted that ischæmia is one of the most potent causes of unbearable pain. I was reminded of this fact during recent years in Japanese prisoner-of-war camps. The gangrenous ulcers of pellagra and malnutrition were the cause of great misery. For the latter excision of gangrenous tissue often promoted healing, but frequently high amputation was necessary. Healing comes from within. Rest and feeding are prerequisites to successful surgery.

Refrigeration anæsthesia is an advance. Four of my amputations last year were done by this method and caused no inconvenience to the patient.

There are many examples of the failure of the healing processes being accompanied by intense pain. Compare the "dry socket" so well known to the dentist, and the pain of gas gangrene. The indolent varicose ulcer on the medial malleolus may be intensely painful. A transverse incision, one inch long, across the middle part of the leg will divide the saphenous nerve and the obstructed saphenous vein and give instant relief, with rapid healing of the ulcer. Of course, routine surgical or injection treatment (or both) of the main saphenous system should be carried out.

Every surgeon knows that while a fracture remains unreduced, vague but real pain persists. Vasomotor changes are seen in the limb. On reduction of the fracture the pain disappears and the vasomotor effects pass away. No amount of physiotherapy will replace the efficient reduction of the fracture.

The strange phenomenon of Sudeck's atrophy is not readily explained. Supporting the limb with Unna's stocking and allowing time to pass—these are measures which usually succeed. I have not found periarterial sympathectomy necessary.

Vascular Lesion—Raynaud's Disease.

The pain of Raynaud's disease is relieved by sympathectomy, but in some cases colour changes recur with the appropriate stimulus—that is, cold. I have used both the posterior and the anterior approach to the stellate ganglion. Ganglionectomy including the second thoracic ganglion is in my experience more certain than trunk section, from the point of view of complete removal of all connexions of the sympathetic trunk with the brachial plexus.

Aneurysm of the subclavian artery requiring ligature of the innominate artery was the cause of intense pain in the arm in one case.

One woman, who had rheumatoid changes in the hands together with Raynaud's disease, was completely disabled. Since bilateral stellate ganglionectomy was performed she has for ten years been able to use her hands normally. However, she has high blood pressure, and recently had a slight stroke. She is now aged sixty-eight years.

Another woman lived comfortably for two years, then died of nephritis.

Is it a disease entity or a phase in a degenerative vascular condition? One elderly patient ruptured a calcified subclavian artery on the fourteenth day after sympathectomy.

Low Back Pain.

Low back pain and sciatica have been discussed freely of recent years. Not all are of skeletal origin. Some are visceral. If each case is considered on its merits, occupation, posture and the vulnerability of certain parts of the spine (cervico-thoracic, lumbo-sacral) being remembered, it is less difficult to put one's finger on the real trouble. The vertebral foramen through which a spinal nerve emerges is a danger spot. The pain of tuberculous spine, of spondylitis and of lumbo-sacral arthritis is relieved by adequate splinting until healing or ankylosis occurs. Bone grafting may be necessary to stabilize the part. Weakness of the supporting muscles or loss of tone in the *erector spinæ* may permit alterations in the relations of the vertebræ and produce back pain or even radiating pain in the legs. Septic foci (not so fashionable nowadays) should not be forgotten. Persisting root pain, so-called radiculitis, such as sciatica, brachial neuritis, requires investigation. The vulnerable areas should be examined—that is, the intervebral foramina and other regions where compression of nerve trunks may occur, as at the thoracic inlet. Fascia, ligaments, disk and articulations and osteophytes may contribute to the compression of a nerve. Aggravation of the pain on coughing or straining at stool usually indicates a foraminal or intradural lesion. X-ray films will show the foramina, if taken at the correct angle.

During the past year I have treated a number of patients with radiculitis of the brachial, lumbar and sciatic regions. Some whose pain was due to spondylitis have been rested in a suitable brace. Some, with pain probably of disk origin, have been rested in bed for a month. Others, whose pain has failed to subside with rest, have had the appropriate foramen explored or a protruding nubbin of disk removed, or if the whole nucleus was degenerated, then it was extirpated. This applies to the lumbar region only; a cervical disk cannot easily be removed, and I have been content to nibble away bone and cartilage to enlarge the foramen. In two cases the lumbar disk was diffusely swollen, and as the nerve was seen to ride gently over the disk margin, nothing more than decompression of the foramen was done. In one case a meningioma filled the fourth cervical foramen and extended along the nerve. This patient had been having physiotherapy and osteopathy for three years.

In four cases the lumbo-sacral area was fused by bone graft. In one case the sciatica had been treated elsewhere by manipulation; but X-ray examination and biopsy revealed a malignant secondary neoplasm pressing on the first sacral root. It is of interest to note that fifteen years ago in most cases we treated severe sciatica by fixation. The results were as good as at present, but more time-consuming.

In three of my own cases manipulation was tried; it was successful in one, and the other two patients came to operation. Although a ruptured disk with pulp herniation is a common cause of nerve compression, it is not the only cause. Undoubtedly it is the slippery element bounding the foramen, and dramatic results follow the simple operation of its removal; but articulations should not be forgotten. Cartilage elsewhere in the body is known to shrink when the peculiar reaction to injury has subsided.

Severe low back pain and sciatica due to inoperable pelvic malignant disease are sometimes a problem.

On several occasions I have injected one or two millilitres of alcohol into the spinal theca, with the patient in the lateral Trendelenburg position. It is a merciful measure and gives complete relief, and may not cause paralysis of sphincters and leg muscles. If it does, it is of no great importance in such cases. One such patient continued working about the house until the day before she died, free from all pain. Another man in a Japanese prisoner-of-war camp had a sarcoma of the *os coxæ*. Pain was completely relieved and urine was expressed from the paralysed bladder. Chordotomy is of value in such cases; but when debility precludes such an operation, the intrathecal injection of alcohol will stop the pain. Why not completely relieve the patient of the intolerable pain?

In ascending traumatic neuritis and painful amputation stumps and causalgia, if vasomotor phenomena are in evidence—sweating *et cetera*—sympathectomy is well worth a trial. If no such phenomena appear, the less surgery, the better. Chordotomy may be necessary, but is not in my experience always permanently successful. The pain has other pathways than the spino-thalamic tracts—for example, Lissauer's tract. Hence the operation of commissure section.

Of the value of leucotomy and parietal ablation in these conditions I have no first-hand knowledge. Intrathecal exploration will be necessary when the presence of pachymeningitis, meningioma or some other tumour is suspected and confirmed by myelography. In the few cases of this kind, one or two a year, in which I have given this treatment, the results have been most gratifying.

Pain in the Upper Extremity.

Pain in the upper extremity may be caused by a variety of lesions. The fascia may be involved; fibrositis or local conditions such as supraspinatus tendinitis may be present, in which case "Novocain" injections frequently bring relief; or if the condition is severe, as in two of my cases of calcification of the supraspinatus tendon, uncapping of the shoulder joint by division of the acromion and removal of the diseased tissue may be required. A painful spot over the scapular notch may be injected with "Novocain" and saline solution. The brachial plexus may be compressed at its emergence from the spine—that is, a foraminal lesion may be present, as described above. Another site is the thoracic inlet, where the *scalenus anticus* may compress the third trunk of the plexus and the subclavian artery. Adson's test is of value.

A typical history is that of a wharf-labourer, who complained of severe pain in the forearm and hand. Wasting of the small muscles of the hand and paræsthesia and patchy anæsthesia in the hand were present. He slept with his arms above his head. (This is the story in four of my cases last year.) At operation under local anæsthesia, the moment the *scalenus anticus* was divided, he said: "Ah! The devil has gone!"

The cervical rib, if present, may not require removal; the decision will depend on its size and its part in causing the compression. In milder cases of this kind the condition may respond to physical training (elevating the shoulder girdle) and proper posture.

The ulnar nerve at the elbow may be chronically inflamed as it passes around the medial epicondyle, either (i) because of old injury to the bone, or (ii) because of trauma from stretcher pressure. During the war I transplanted the ulnar nerve in patients who during long recumbency on bamboo benches had developed this condition.

In the past year four such cases have been dealt with in our clinic. The results have been very good.

The median nerve may be compressed at the wrist beneath the transverse volar carpal ligament. Two such patients presenting the usual pain, not confined to the hand but vaguely spreading over the forearm and arm, associated with wasting of the thenar eminence and anæsthesia of median type, were operated upon last year. Division of the volar ligament brought complete relief from pain.

Pain in the Face and Head.

There are four or five well-defined types of pain in the head which are referred to the surgeon.

The first group consists of organic lesions—brain and dural tumours, aneurysms, nerve tumours, meningo-vascular syphilis, post-meningitic lesions, bone disease, inflammation and neoplasms, special sense disease (eye and ear) and unerupted teeth. I shall not refer to these. Usually there are signs—cranial nerve palsies and other evidence of disease—besides the pain. The treatment is that of the lesions in question.

The second type comprises pain referred to the head from cervical spine disease, teeth and antra, post-herpetic neuralgia, malignant disease, and pain referred from tongue and pharynx.

The third type includes pain of periodic nature—migrainous neuralgia, ciliary neuralgia.

The fourth comprises facial neuralgia of doubtful origin.

The fifth type is paroxysmal neuralgia—*tic douloureux*. In the second, third, fourth and fifth groups I have had over 200 patients referred to me in the past twenty years. The treatment of post-herpetic neuralgia is not settled. Section of the sensory root of the fifth nerve and alcohol injection of the Gasserian ganglion have both failed to cure it in my hands. Repeated injections of "Novocain" and saline solution into the painful skin areas help. For the pain of radio-necrotic ulcers of the tongue and mouth nerve section and alcohol injection of the Gasserian ganglion have provided relief. Usually the ninth nerve needs to be cut. Relief may not be complete, but the patient is grateful for small mercies. I have also divided the cervical plexus in cases in which the glands have been irradiated and pain persists.

I mention two severe cases of migrainous neuralgia which resisted medical treatment and caused major incapacity. Both patients were referred to me after long and painstaking investigation and many forms of medical and endocrine treatment.

A woman, aged thirty-five years, in 1935 had suffered severe right-sided migrainous neuralgia for several years. In and out of hospital, under many specialists and two of our Royal Melbourne Hospital senior physicians, she had become a "cot case". I injected the right Gasserian ganglion with 0·5 millilitre of absolute alcohol, producing anæsthesia of the whole right fifth nerve field. Care was taken to protect the eye. The neuralgia was completely cured. Ten years later the same kind of pain occurred on the left side of the head and face. Deep X-ray therapy to the ovaries was applied by Dr. R. Kaye Scott, and produced complete relief. The menses ceased, and the usual menopausal symptoms set in. These did not worry her unduly. In December, 1947, she was a healthy, happy woman, but stated that slight sensory return on the right side of the face had recently occurred.

The second patient, a male, aged thirty years, was invalided out of the services in 1944 with severe right-sided migrainous neuralgia. All investigations gave negative results. The pain involved not only the right side of the face and head, but the occiput and the right side of the neck as far down as the third and fourth cervical segments. Injection of the Gasserian ganglion with alcohol completely relieved the pain and enabled him to resume normal employment.

The distribution of pain to the occiput can be explained by the spread along the *nervus tentorii*, a branch of the ophthalmic nerve; but the cervical radiation is difficult of explanation unless one invokes the theory of Ross and Mackenzie. The descending root of the fifth nerve passes down the cervical cord for several segments. Supraorbital neuralgia, sometimes mistaken for *tic douloureux*, is best treated by endocrines or, alternatively, Gasserian ganglion injection. Avoid surgery.

In the fourth group I place a number of vague, constant, dull, aching forms of pain in the face, particularly met with in women. Some years ago most of the patients had had teeth, sinuses *et cetera* treated surgically. In some cases the pain was due to persisting bone disease, the result of infection and operative trauma. In others it resembled amputation stump neuralgia, and perhaps fibrils of nerve were caught up in scar. The theory of Sluder was invoked. In one case I removed the sphenopalatine ganglion, with a negative result. In many cases there were obvious psychological factors. In the late 1920's I was persuaded to treat three of these patients by fifth nerve sensory root section. No relief was obtained. The fifth nerve is not the only sensory nerve involved. The seventh nerve and the autonomic nerves also probably transmit pain impulses from the damaged facial structure. During the past fifteen years I have treated such patients with a minimum of surgical interference—perhaps local injection of the infraorbital or the spheno-palatine nerve, perhaps avulsion of the infraorbital or the supraorbital nerve. The main elements in treatment are reassurance, suitable diet with extra vitamin B_1, sedation and the promotion of improved general health, and endocrine correction. A little psychotherapy may help, provided it is of the simple, confessional method. The tongue is sometimes mightier than the scalpel.

True Major Trigeminal Neuralgia.

Over 150 patients suffering from trigeminal neuralgia have been referred in the last twenty years. The paroxysmal pain, commencing in the fifth nerve field (occasionally the ninth nerve, in one case the seventh nerve and in two cases occipital), associated with chewing and talking, accompanied by lachrymation and salivation, lasting for seconds only—terrific, lancinating like red-hot needles and electric shocks—presents a picture which once seen is not soon forgotten. Most patients are edentulous, having lost their teeth or had them extracted in the hope of obtaining relief. The treatment of this condition is well defined. The diagnosis is easy and there should be no passing of the patient from one specialist to another in the vain hope of discovering a cause. There are no signs, but the symptomatic picture is clear cut. This does not mean that a careful neurological examination is unnecessary, nor should a general assessment of the patient's medical status be neglected. In minor cases local injections of a few drops of absolute alcohol at the appropriate foramen (mental, infraorbital or occasionally supra-orbital) will give temporary relief. I have seen two patients with bilateral trigeminal neuralgia. The disease is more common in the professional classes, hence the small series at the Royal Melbourne Hospital.

As many of the patients were elderly and suffered from other diseases (arteriosclerosis, cardiac disease *et cetera*), and frequently were in poor physical condition from long starvation, I have used the Hartel injection of the Gasserian ganglion.

In my last 128 cases injections were given in the manner described. Results have been on the whole good. Thirty-six patients had a repeat of injection or operation in periods varying from one to ten years. The Harris lateral approach has been used only for younger patients suffering from neuralgia in the mandibular division only, and then it is pointed out that fractional section of the nerve root may be necessary later.

In 10 cases out of the 128 section was performed either (*a*) because the lower division had been injected once or twice elsewhere (six cases) or (*b*) because of recurrence after ganglion injection by me or failure to introduce the needle through the *foramen ovale* (four cases). Five other patients were treated surgically at their request or because of the need to avoid eye complications.

In early days I had some failures and disappointments both in injection and in section, but with experience these have been avoided. The Harris method of extracranial injection of the mandibular nerve is relatively easy. The intracranial ganglionic injection calls for patience and skill. The latter injection is given under local anæsthesia

with the patient sitting in the consulting room or at home or in hospital, as circumstances permit. The details of technique need no elaboration; suffice it to say that I rely on the feel of the anterior margin of the *foramen ovale* and slip the needle through gently for a quarter of an inch, frequently withdrawing the piston of the syringe to see that there is no cerebro-spinal fluid or blood. The patient's reaction is sufficient to indicate the site of the needle point, and the description of the spread of pain tells which part of the ganglion is penetrated. One drop of alcohol confirms this finding; it is followed by four or five drops, which require some pressure to inject, until the face begins to redden and perhaps the eye to become pink. Fractional injection has been done purposely on many occasions, an attempt being made to spare the ophthalmic part of the ganglion. The relief of pain is immediate, and a formerly tearful, distressed patient rises from the chair with a smile, the first for many months.

A cotton suture draws the eyelids together. Eye drops are prescribed and the patient is told that a crop of herpes will appear about the mouth in a few days. Two days later, if the eye shows no sign of trouble, glasses with a lateral shield are ordered to be worn for six months. This was necessary in 22 cases. If the eye is red and corneal softening is imminent, the lid margins are scarified and sutured carefully by myself or by an oculist. This latter safeguard has been necessary in four cases in this series and in one case after section of the root. It is to be noted that the last patient had had elsewhere much alcohol injection without complete success.

Occasional trophic changes affecting the cartilage of the nose on the affected side have been noticed (two cases).

Why not cut the nerve in all cases? The statistics given below, showing amongst other things the age of the patients, indicates that radical surgery should be avoided if possible. If a simpler method succeeds, why not use it? Admittedly there are a few patients who are not cured by injection for a variety of reasons. Four such patients were operated on by me in the last year—three because of "failed injections" (two of my own), and one because multiple injections extracranially prevented the entry of the needle. This man had high blood pressure, was in bad condition, and died two days after operation. He took his anæsthetic badly and at autopsy presented a number of reasons for his failure to survive. On the other hand, 20 other nerve sections preceding his were successful, one patient being a woman, aged seventy-eight years.

The following statistical information applies to 128 patients with *tic douloureux* (fifth nerve) in private practice. The age distribution in decades was as follows: fifth, 30 patients; sixth, 15 patients; seventh, 45 patients; eighth, 31 patients; ninth, six patients; tenth, one patient. The whole fifth nerve was affected in 100 cases, the mandibular division alone in 15 cases, the maxillary division alone in 13 cases and the ophthalmic division not at all. Five patients who had ganglion injection subsequently required operation. Section of the root was carried out on 15 private patients and on six patients at the Royal Melbourne Hospital. Eye complications after injections were estimated as follows: 22 patients required suture of the lids up to two months, and four required it for six to twelve months. There were 36 recurrences; further injection was carried out in 31 cases, operation in five. In the whole series one patient died; operation was carried out at the Royal Melbourne Hospital.

In a previous series at the Royal Melbourne Hospital, not included in the foregoing, an unspecified number of patients were treated between 1927 and 1935. It was impossible to obtain their records. Many were treated by injection in the out-patient department. There were three post-operative deaths, one from pontine hæmorrhage, one due to anæsthesia (chloroform) and one from cavernous sinus hæmorrhage (this patient had recently undergone injection elsewhere).

The risk of operation is real, though small, and I consider that in the elderly the ganglion injection is safe and produces sufficiently lasting effects to warrant its trial. For younger patients fractional section is ideal, after extracranial injection has ceased to have effect. In "failed injection" cases complete posterior root section is the only satisfactory treatment.

The operative approach and instruments used are according to Adson's technique. In two cases I have used Dandy's posterior approach because it was desirable to explore the cerebello-pontine angle. One of the patients died from hæmorrhage.

Many of my old patients treated by injection fifteen or more years ago keep me informed of their freedom from pain, and some regularly send their greetings on the anniversary of their release from torment. Perhaps this is the most satisfying reward a doctor can receive. At the expense of being platitudinous, I would remind you that among our multifarious exertions on behalf of suffering humanity not the least is the relief of pain.

MASTOIDITIS IN INFANCY.

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THIS is a review of 53 cases of mastoid disease in infancy, in which from July, 1946, to January, 1948, operation and/or autopsy was performed at the Perth Children's Hospital, and of a larger unknown number of similar cases which had neither operative nor post-mortem verification.

Arbitrarily the age of the group is considered as up to two years; most of the patients were aged under fifteen months; 44 patients, of whom 38 were operated on, had "latent" mastoiditis and nine had "frank" mastoiditis with local signs. The "latent" cases proclaimed themselves by general (especially gastro-intestinal) symptoms and a minimum of local signs. In these, operative decisions were made on general, not local, considerations.

LATENT MASTOIDITIS.

Before this series we were only groping our way among cases of latent mastoiditis at this hospital. The basis of our recent approach was the old pædiatric teaching that, in infancy, refractory alimentary tract conditions (persisting with no proof of any specific bowel infection, or of any of the classical pædiatric diseases) were probably due to a hidden septic focus. To this we added that with modern hospital facilities of investigation the hidden focus was not likely to be pneumonia or pyelitis. So middle ear conditions remained of the old "pyrexia of unknown origin" triad.

In the period immediately previous to this series, the hospital had admitted a number of infants, ranging in age from four weeks to fifteen months, failing to thrive, feeding with difficulty, having over-frequent bowel actions and general weight deterioration, with exacerbations of all these symptoms. These infants were seldom acutely ill, but the whole picture was progressive, and provisional diagnoses of "enteritis", "marasmus", "liver abscess" and "pyloric stenosis" were carried for one to three months till final death from dehydration, debility and toxic circulatory failure.

At the post-mortem examinations, except for signs of final circulatory failure and terminal lung infection, nothing of note was found except a low-grade infection of mastoid cavities. About the same time a few patients who had early shown some signs of acute *otitis media*, and in whom repeated examination had revealed thickening and redness of the postero-superior quadrant of the *membrana tympani* (causing a "forwardness" of the membrane which obscured the rest of the membrane by increasing its obliquity), were submitted to mastoidectomy under local anæsthesia as a last resort, and recovered.

For the eighteen months under review such cases were regarded as probable cases of latent mastoiditis, and were tackled more thoroughly. The results in these latent cases were as follows: 38 patients were operated on, and practically all had bilateral mastoiditis; eight patients

died, mostly from other causes, and mostly after the operation area had healed.

Half-castes of Australian aboriginal blood (not other half-castes) did badly and died. Their infection of the mastoid cavity seemed to go on quietly spreading after operation, and post-mortem examination showed that infection of other parts had behaved likewise (for example, the lungs had had successive non-resisted lobe infections).

Most of the other subjects were poor type babies from foundling homes with other lesions; two had hare-lips and cleft palates and several had congenital heart disease. Many had a history of difficult delivery. A few were unexceptionable in family and birth and nursing histories. One was a doctor's child.

At the same time, for each patient operated on, approximately four patients with the same signs and symptoms were watched, and gradually recovered. Operation was not undertaken if, with chemotherapy and/or penicillin therapy and intravenous supportive therapy, the child was holding its own in general health; operation was not even considered if the child's condition was improving and if it had general good tonus, bright eyes and good skin, and was not repeatedly dehydrated.

The main decision, with the help of the physician, was made purely on general grounds, in the absence of any other proven diagnosis, and in view of the failure of repeated chemotherapy and penicillin therapy and intravenous therapy to halt the downward progress. Bilateral mastoidectomy was performed as an emergency measure on several such patients, who in the opinion of the senior medical staff were moribund.

C.H., a baby of mixed (Italian) blood, had had poor early care and was illegitimate. He was admitted to hospital on December 16, 1946, with the provisional diagnosis of "pink disease". He went steadily downhill, with vomiting, lethargy and bronchopneumonia. His condition was improved by blood transfusion, and then it grew worse, with increased vomiting and diarrhœa, in spite of fresh intravenous therapy. Operation was undertaken as an emergency measure on February 6, 1947, immediately after the first consultation, because the child looked unlikely to live till the next day; his age was one year, his weight was nine pounds and his appearance was corpse-like. Both mastoids were found full of pus, from which *Staphylococcus aureus* was isolated. He steadily and gradually recovered in spite of extensive lung infection at the time of operation.

In pre-war days, with the absence of laboratory-controlled intravenous therapy, patients with such advanced infections would have been bound to die, and operation would have been unnecessary cruelty. In the 1930-1935 period these cases were much under discussion; but then, if they were left as late as the present series were, for the diagnosis to be certain, the patients died. If they were operated on earlier and successfully, many must have been in a group corresponding to that in the present series in which operation was seriously considered, but abandoned on improvement with intravenous therapy.

Factors Influencing the Decision about Operation.

Other factors influencing the decision about operation were as follows.

Operation was undertaken if the infant had already been tided over two or three "flare ups" under difficulty, and if the changes of next "flare up" were likely to be irreversible.

If the infant showed signs of inflammation—generally subacute—in the nose, pharynx or naso-pharynx, operation was at least postponed, and these conditions were considered as a likely cause of the current general deterioration. If the postero-lateral part of the *membrana tympani* and the external auditory canal showed congestion in the absence of the above-mentioned nose and throat inflammation, this picture was considered significant, as suggesting blocked residual infection in the mastoid.

The appearance of the *membrana tympani* on repeated examinations was taken into consideration. As a general rule, the patients who were admitted to hospital with frank *otitis media*, and in whom perforation or incision had productive results, recovered (these infections were often labelled "early mastoiditis"). The patients with latent mastoiditis had a slight history or no history of *otitis media*. Their early notes sometimes mentioned a redness of the *membrana tympani* which seemed to have cleared up. The membrane's appearance was treacherous and required frequent watching; care had to be taken not to obscure the canal by trauma during the examination. The most significant finding after the early flush had subsided was a persistent redness of the postero-superior quadrant extending onto the neighbouring canal skin, but not a true sagging of the posterior canal wall as in adult mastoiditis. This segment was often mistaken for the *membrana tympani* completely swollen. It obscured the rest of the *membrana tympani* partly by making the obliquity of the anterior and inferior three-quarters more horizontal (more "recessed"), and partly by filling the tip of the tiny speculum needed in the examination of such poorly developed infants. When it was possible to bring it into the line of vision, the major part of the *membrana tympani* appeared normally grey and translucent. On incision of this postero-superior area pus was seldom obtained; if any was obtained, it was just a bead which did not keep flowing; but such a result was valuable evidence of obstruction and infection. Careful incision after packing with adrenaline to prevent obscuring was necessary to get this evidence. Sometimes a *membrana tympani* which had aroused no suspicion and had not been seen to be red, showed thick and white and nearer the speculum than at the previous examination, and on incision frank pus was obtained, which did not continue to flow the next day.

Baby P. was aged five months. The child had good living conditions and healthy parents. Mastoiditis was frequent in various branches of the family. The child had had attacks of dietetic upset associated with ear pulling. A fresh attack, similar but more severe, had lasted for two days. The left *membrana tympani* was congested in the postero-superior region. Incision produced pus, with lessening of the symptoms. These recurred, and five days later the right *membrana tympani*, which had been normal, though still pale, was nearer the observer. Incision produced pure pus, the flow continuing for twenty-four hours only. The child's condition improved. Then the patient contracted a bowel infection during a Salmonella outbreak, and fresh ear incisions were unproductive and intravenous therapy was unavailing. Double mastoidectomy was performed and a gross spreading infection was found. With further intravenous therapy the child recovered, in spite of further attacks of Salmonella infection; no further mastoiditis occurred.

In other words, in this case the mastoid infection, which had been secondary in its incidence, later became a primary source of systemic toxæmia, isolated and shut off from drainage into the middle ear, because of swelling about the cavities and folds, and attachment of the ossicles. Thus was caused the factor next to be discussed in relation to the decision about operation.

In cases of "failed repeated myringotomy"—that is, when incisions had produced pus at first, the flow of which had ceased with continuance of general symptoms—it was decided that it was better to perform mastoidectomy than to make a third incision, on the presumption that drainage from the aditus and attic into the middle ear proper, and thus through the incision, was blocked.

A general rule was: "If in doubt, wait and see." This was not always possible if the child was thought to be dying. The cousin of Baby P. (see above) had similar attacks without Salmonella infection and gradually recovered.

On the other hand, among the post-operative deaths, two of the patients had been operated on whilst the first acute phase of toxæmia was present, though subsiding; our hands were forced because of fear of death during the first phase. At operation and autopsy, one of these children was found to have mastoids with very small spaces, and the surrounding bones were actively infected. Presumably the child was handicapped, as unlike most of our patients he did not have large mastoid spaces to act as reservoirs whilst the infection lost its virulence. The other was a baby, aged only five or six weeks, who seemed to be going rapidly downhill in the first phase of infection. The child was grey and ill, had several attacks of circulatory collapse, and died five days after operation. If we could have waited longer until the next "flare up", the child's

resistance might have increased and the responsiveness of his peripheral vascular system might have been better by practice against variation. The well-known instability of water balance in young babies was associated with these factors. Children aged over six months or even three months, though looking dreadfully ill, did better than the very young. (Baby C.H. at one year weighed nine pounds, and had extensive pneumonic infection. At operation both mastoids were old wells of pus, yet he responded well. Younger babies, not so dehydrated but "grey looking", failed to pick up.)

The leucocyte count was helpful, especially if investigation proved the absence of other infection (for example, of tonsils, chest or renal pelvis). Generally a moderate leucocytosis was present, which disappeared in patients left until moribund.

X-ray examination was of some help, but generally sedation was needed. In such frail patients even this amount of sedation, with a period of dry mouth, cough, aspiration of mucus *et cetera* and lowered circulation, was harmful. X-ray examination helped if it showed: (*a*) that the mastoid cavity was large or loculated widely for the age of the infant and therefore a potential reservoir for quiet infection; (*b*) that the semi-circular canals stood out so clearly as to suggest destruction of the neighbouring cortical walls. A case of tuberculous granuloma involving the external and middle ear and mastoid showed this very clearly (Law, 1920).

The decision whether and when to operate was generally made by the physician when other diagnoses had been excluded, when the patient had been left as long as was considered safe, and had been temporarily "pepped up" as much as possible by intravenous therapy without any "flare up".

Operation.

Anæsthesia.

Local anæsthesia was used; 1% or 2% procaine ("Novocain") solution with four minims of a 1 in 1000 adrenaline solution per ounce was employed. Generally less than one ounce was needed to complete the procedure on both sides. The solution was injected with a fairly fine needle at the usual sites to block the nerve supply, and under the periosteum, and especially down and forward to try to flood under the periosteum at the region of the posterior canal wall (a finger in the meatus gauged this). The local anæsthetic solution was massaged in firmly. The baby was "mummied" on a cross in the usual way (as for Ramstedt's operation under local anæsthesia) and was given a small pre-operative dose of "Seconal" sometimes (see above note concerning sedation), occasionally a little "Pentothal" in the intravenous drip fluid, and generally some brandy and water to suck.

The adrenaline seemed to bolster up the infant over the operation; but all patients were "pepped up" as much as the medical registrar thought possible just before operation by controlled intravenous therapy. (I had a liking for serum before and after operation, remembering our disasters of pre-serum days). Likewise such controlled intravenous therapy was very important after operation. If it was not adequately and correctly carried out, the patient, after seeming well immediately after the operation, collapsed badly about thirty-six hours later. This was a very common fate ten years ago.

In this series with eight deaths the time of death after operation was as follows: two to three months, three deaths; one week, three deaths; two weeks, one death; two days, one death.

Technique.

Various incisions were tried, but approximately the best was one straight up from where the mastoid tip should be (without any curve forward of the lower end, as the facial nerve emerges laterally and superficially in these infantile tiny mastoids with undeveloped tips). The top of the incision curved in practically to the external ear. Even with this upper curve forward the incision seemed high above the canal (which at this age is near the bottom of the external ear), and one had to elevate down and forward about one inch to expose the posterior superior wall of the bony canal (no spine has developed at this age, so without this landmark topography is difficult). The usual incision through the periosteum was made, and at its upper end a little horizontal nick was put in the periosteum; otherwise the elevation tended to wander up under the temporal muscle. The periosteum was elevated down and forward with a heavy (Farabeuf) elevator or with gauze on the thumb. (The periosteum peels well only at the site of operation, and sharp elevators dig into the green bone too much.) A small retractor was then put in and the mastoid area was "uncapped" by shaving off the surface of bone immediately above the posterior superior bony canal margin with a hand gouge. In some cases pus "popped" out; in others (the majority) the cavities were found filled with thick pus, general debris, granulation tissue and spoilt mucosa. In each case except one the mastoid cavity was large in proportion to the age and size of the child. In many cases, even when the baby was only a few months old, there were extensive true cell ramifications; in others there were warrens of spread of infection in bone substance simulating true cells. These cavities (either type) were guttered out gently with curette and *rongeurs*, and part of the posterior bony canal walls was nipped out to prevent forward droop of the external canals. The wounds were packed with pure penicillin (after delay and gauze packing to achieve hæmostasis, so that the penicillin should not be washed out by blood), and then sutured around fine twisted glove rubber drains. Such a drain gave enough "squelching" movement to allow discharge (generally slight) without inviting external contamination, and it also seemed to prevent the "flopping in", and/or attachment to the bony margin of the cavity (often large) of the anterior skin edge. The wounds were sealed with small dressings; the traditional large mastoid dressing, if used for these tiny babies, pushes forward the external ear, thereby causing "jug handle" auricles.

The rubber drains were half-way removed in thirty-six hours and completely removed after seventy-two hours. Most of the wounds healed well, unless too much antiseptic solution was used on an apparently moribund baby.

Operative Observations.

In some cases the side thought before operation to be the worse was the less infected, though sometimes the more vascular, whilst the "quiet" side was a cess-pool of pus, and by its hard sclerosed walls showed that it was obviously of longer standing. Therefore, after attempts to treat only one side in early cases, this method was abandoned and bilateral operation was performed. (To operate on the second side when the patient is prepared does not take much longer.) Twice, after little abnormality had been encountered on the first side, in the second pus under tension was found.

In one early case the patient (J.E.) was found to have pus on the side under suspicion, and on this side only was mastoidectomy performed. This child's condition improved, then she developed varicella and became gravely ill. Myringotomy on the other side produced pus; the flow of this soon ceased and the baby's condition became worse. The second mastoid was opened, and gross, tense pus was found in an old, sclerosed, infected cavity.

Another patient (R.J.) underwent mastoidectomy on the suspected side in December, 1946. His condition improved for a few days; then he resumed his "toxic" look, and resented, not the dressing of the wound, but any approach to the intact side. Operation on this side was carried out as emergency on December 24, 1946, and an infection of long standing with spread into bony tissue was drained. Rapid recovery followed.

As has already been mentioned, in practically all cases the cavity was large and multilocular, often as large and branched (whether by development or by disease) as that found in the average two-year-old. The mastoid antrum, as is usual in infants, was high, but nevertheless, after infection from the middle ear, appeared to be blocked at the aditus, and to have become the primary source of the general toxæmia. The following factors seemed integral to this result.

1. The infection was of low grade, with plenty of space in which to spread, and producing not very fluid pus, which

therefore stagnated. (Laboratory investigations on pus obtained at operation often revealed no growth, *Staphylococcus aureus* or pneumococcus, and occasionally a hæmolytic streptococcus.)

2. The mastoid antrum, though high, was large enough to act as a branching dependent reservoir of infection below the aditus. It was like a pear with the body hanging down a little from the postero-superior region, the aditus representing the stalk. This stalk (aditus) was naturally narrow at this age, and was increasingly narrowed by swelling of its own lining.

3. The exit from the attic to the middle ear was further blocked by inflammatory swelling of attachments and mucosal folds or the ossicles; it is to be remembered that in infants the ossicles are practically adult size, whilst the spaces of the aditus and attic are tiny. In many long-standing cases the infection was eating into the marrow of the petrous bone rather than forcing itself externally through the cortex or returning into the middle ear. All these conditions combined to produce a latent mastoid infection acting as a primary toxic source after the *otitis media* had subsided. Further, in some cases the infective process had ceased on one side, leaving a hard sclerosed cavity. In view of this, and since many patients recovered without operation, one thinks that the frequent X-ray appearance of sclerosed mastoids in later life is due to early latent infection. Wittmark elaborated this theory years ago.

Duration of Operation.

Operation on each side took twelve to twenty minutes (depending on the extensions of the infection); so with the injection, the changing over and "scrubbing up" between the two sides, the total was about an hour. Most patients seemed to stand it well; but this is no reason to subject a sick baby to such trauma unnecessarily. In cases in which only little infection was found and the baby was very ill, operative trauma prejudiced life; but when gross pathological change was found, the benefit done outweighed the disadvantages. Some patients immediately ceased vomiting and began to pick up; some successfully withstood fresh ward infections (bowel and upper respiratory tract); see the comments on baby P.

Post-Mortem Findings.

There were fourteen deaths; eight of the patients had been subjected to operation and six had not. The general finding was peripheral vascular circulatory toxic failure, especially evident about the brain, where presumably the toxic spoiling had become irreversible. Various subjects had infective lesions of the lungs, bowel *et cetera*, and toxic spoiling of the heart, liver *et cetera*. In the six subjects not operated on, thick pus and granulation tissue and bony inflammation of considerable duration were also found in the mastoid cavities. The fifth autopsy of this group was one which I attended to secure the temporal bones for lecture purposes. The general findings were only as above, and non-specific, and the history was of the type of the cases under review; so the pathologist asked me to open the mastoids there, and both were found to be large and packed with "mushy" infective granulation tissue. The sixth subject was one who had been admitted to hospital several times as "dietetic", and discharged from hospital "improved". The child was readmitted to hospital suddenly, severely ill, and soon died. Autopsy revealed general toxic changes as above, together with gross purulent infection of both mastoids. Latent mastoiditis had not been considered previously. The other four subjects presented the same pattern, but at the beginning of the series, and were the basis for consideration of more frequent operation.

The following are notes on the eight patients who died.

Baby W. died three months after operation. He was of Indonesian blood, and had a general low-grade infection of the lungs and bowel.

Baby D. died two months after operation. He was an Australian aboriginal half-caste. He had had successive recurrent large patches of pneumonia, and renal hæmorrhage. He also had recurrent infection in the petrous bone and a Salmonella bowel infection.

Baby C., who died two months after operation, had a practically identical post-operative course and similar post-operative findings to those of baby D.

Baby L. died two weeks after operation. Autopsy revealed patchy hæmorrhagic areas in both lungs and gross wasting.

Baby Wa., who died one week after operation, was one of the very young babies mentioned as "poor risks". Pus under tension was found at operation. The baby died suddenly from acute circulatory toxæmia and failure.

Baby T. died one week after operation. His condition improved immediately after operation; then he developed agranulocytosis and skin and mouth infection. He had had a heavy course of sulphaguanidine for a Salmonella infection. Autopsy revealed complete absence of lymph glands (even mesenteric) in spite of the bowel infection, with general toxæmia and lung infection.

Baby N. died one week after operation. He had suffered from pyloric stenosis; both medical treatment and operation had been failures. No ear signs were found to justify operation, and the baby was vomiting blood at operation. Autopsy revealed wasting, dehydration and circulatory collapse.

Baby Li., the other very young baby mentioned in this paper, died two days after operation. Autopsy revealed infected tissue in both mastoid operation cavities, purulent bronchitis, and atelectasis. The suspicion of fibrocystic disease was aroused.

Literature.

I had not much access to the literature on this subject till nearly the end of the series; but approximately the same conditions and views are expressed in articles by Burns and Gunn,[1] by Leathart,[2] and by Patterson and Smith.[3] Jackson[4] makes the following statement:

> At birth and during infancy the mastoid Temporal domain is filled with primitive lipoidal bone marrow. This is completely replaced by pneumatic cells by the third year. Consequently an inflammation of the mastoid during infancy is an Osteomyelitic process, while after the 3rd year it is an Osteitis. In the presence of an infection of the mastoid in an infant, the outstanding symptoms, in addition to the Otitis, may be severe constitutional disturbances, especially diarrhœa, because the Gastro Intestinal tract, not having acquired its full functional growth, is in an unstable condition. In an infant the usual subperiosteal mastoid abscess resulting from a suppurative Otitis Media is an ostemyelitic process. The abscess is situated above and slightly behind the auricle because of the position of the tympanic (mastoid) antrum. When not evacuated the abscess has a tendency to spread forward under the temporal muscles.

Non-Latent Mastoiditis.

The latter part of this quotation is well illustrated in the second group of this series—namely, the cases of non-latent or frank mastoiditis. These children were mostly from nine months to two years old, and proclaimed their mastoiditis, not by constitutional symptoms, but by local signs of swellings about the ear, atypical in nature compared with those of older children.

There were nine patients in this group. They were mostly older than the patients with latent mastoiditis, were not constitutionally ill, and had been observed to have an upper respiratory tract infection with *otitis media*; they were strong infants with well-developed mastoids. After the first symptoms, they appeared to be progressing well, and then presented with dramatic ear changes, such as facial paralysis, or subperiosteal swelling; in several instances the swelling was "zygomatic", but was not due to the presence of zygomatic cells; it was due, as was stated by Jackson, to the high position of the mastoid antrum. After dehiscence, high owing to this position, the pus had a "watershed" spread forward due to the nature of the attachment of muscle and periosteum to the zygomatic root. (At operation in latent cases, as has been pointed out, unless the periosteum was nicked transversely and elevated downward and forward over the smooth area just behind the bony auditory canal, the elevation tended to stray up among fibres of temporal muscle.)

One patient (K.) had been admitted to hospital with the diagnosis of mastoiditis, with some post-auricular swelling; this subsided with treatment by penicillin and chemotherapy. The child went home; then he seemed pyrexial and ill and in pain, and was readmitted to hospital. X-ray examination

revealed a deep abscess forming in petrous bone. Operation revealed a healed scar of dehiscence high over the mastoid area, and a deep infection and cavitation in the petrous bone. The area was cleaned and drained, but progress was slow.

This seemed a case of mastoiditis masked by penicillin and sulphonamide drugs; but meanwhile in many other cases early clinical mastoiditis seemed to have been aborted by similar treatment, and similarly, many patients with acute upper respiratory tract infections and early *otitis media* seemed to have been saved from mastoid suppuration by such treatment.

Of the other seven in this group, none had had much, if any, chemotherapy, and that early, and all the manifestations were bizarre—that is, the children had zygomatic swelling, or unruptured *membrana tympani* or sudden periosteal swelling when apparently recovered in general health.

Therefore there seemed to be a high percentage of atypical mastoiditis in this group. This was illusory rather than due to chemotherapy—illusory, because the age group of nine months to two years has always (from causes mentioned by Jackson, together with the presence usually of a large mastoid cavity and a thin cortex with weak suture lines) had a high percentage of atypical cases. These atypical cases are now especially arresting, because the overall number of the classical cases of mastoiditis—especially among children aged two to twelve years—is so much reduced that the patients aged from nine months to two years, with mastoiditis intrinsically often atypical, now form a large, instead of as formerly a small, percentage of the total number of mastoid patients in a children's hospital.

CONCLUSION.

An eighteen months' review of mastoiditis in infancy at the Perth Children's Hospital, Incorporated, is given. A large number of patients presumably suffering from latent mastoiditis recovered without positive diagnosis. Probably their future mastoid development will be impaired.

A large but lesser number (38) seemed to be suffering from latent mastoiditis with general deterioration, until mastoidectomy was performed. Eight of these died, mostly from general causes, not operative or local. The rest of these (30) recovered their general condition steadily and fairly rapidly after mastoidectomy, at which a mastoid infection was found. Generally their post-operative stay in hospital was much shorter than their pre-operative stay. Some examined afterwards in the out-patient department looked chubby and well. Some have had, during fresh upper respiratory tract infections, acute *otitis media* with simple course and recovery.

Mention is made of a small group of similar patients who died without operation, and at autopsy were found to have latent mastoid infection.

A small group of infants, slightly older, but aged under two years, is mentioned, in which the signs were purely local and often unusual, the diagnosis was definite, operation was clearly indicated and often immediate, and pre-operative and post-operative stays in hospital were short. The "latent" group is discussed in detail, in an attempt to evaluate these cases for future guidance in treatment of very ill infants, in the light of modern improvements in intravenous therapy, biotherapy and chemotherapy.

It is suggested (in company with the authors mentioned) that such patients should not be allowed to die without benefit of mastoidectomy.

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I owe great tribute to Dr. H. B. Gill and Dr. H. J. Gray for their constant help and wisdom in all the problems of these cases and of this report. My thanks are also due to the honorary physicians, the medical superintendent and the registrar of the Perth Children's Hospital, Incorporated, for their full cooperation in the anxious care of these babies.

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MEDICO-LEGAL PROBLEMS BASED ON EXPERIENCE AT THE MORGUE.[1]

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Historical Survey.

MEDICO-LEGAL PROBLEMS began in this country with the arrival of the First Fleet in 1788. Collins, the judge-advocate, in his history of the settlement at Port Jackson, has recorded many cases of interest. He gives an excellent account of the death by lightning of two convicts and a dog found dead at the foot of a tree, and his account suggests that they died from asphyxia. Post-mortem examination was practised at the first hospital built on the west side of Sydney Cove—on the site where there is now a morgue. The bodies of those hanged were afterwards publicly dissected—this was an addition to the death penalty, viewed with horror by those about to suffer it. It is recorded that Fenlow, one of the settlers on the Hawkesbury, was hanged for the wilful murder of his convict servant, David Lane, and afterwards his body was publicly dissected and the surgeons found a stone in his gall-bladder the size of a lark's egg. Instead of a coroner, there was a court constituted by the judge-advocate and three naval and three military officers. In another instance, they returned a verdict of justifiable homicide where a drunken seaman was fatally stabbed. Of the first five deaths that occurred when Melbourne was founded nearly fifty years later, four were medico-legal, two were shootings, one was accidental, one was suicidal, and there were two cases of fatal spearing by the blacks. Captain Lonsdale acted as first coroner.

With the growth of Melbourne there has been a steady increase in the amount of work coming within the jurisdiction of the coroner, and now the toll of the roads contributes its great quota.

Alcohol is a great problem and is the cause of much crime and medico-legal work.

Excluding deaths in the mental institutions during 1947, the Melbourne coroner dealt with 1759 cases, and death was due to natural causes in 987. There were 1148 post-mortem examinations and 693 inquests. There were 346 accidental deaths, 86 suicides, 27 fatal abortions, 110 deaths from misadventure, 10 murders, 10 cases of manslaughter, and 58 cases in which an open finding was returned.

Among such material, many medico-legal problems arise. I propose to touch very briefly on a few which are of fairly general interest to the medical profession.

Sudden Death.

Autopsy shows that about 70% of all sudden and unexpected deaths are due to natural causes, and the common diseases are responsible for nearly all of these. About 25% of all the sudden deaths are due to coronary arterial disease. The only common cause of really sudden or instantaneous death is coronary obstruction. Occasionally coronary thrombosis or embolism occurs. Coronary obstruction from atheroma may be followed by infarction and rupture of the heart wall, but this takes some time. Disease of the coronary vessels, disease of the heart and disease of the cerebral vessels predominate. Rupture of the aorta is not uncommon, without aneurysm. It is the ascending aorta that gives way in any part of its

[1] Read at a meeting of the Victorian Branch of the British Medical Association on August 4, 1948.

course between its origin and the arch, and usually rupture takes place into the pericardium. These are really examples of short dissecting aneurysms. This type of rupture is more often seen in my experience than rupture of the heart, which usually takes place on the anterior wall of the left ventricle towards the apex as a sequel to myocardial infarction.

Cerebral hæmorrhage, subarachnoid hæmorrhage from rupture of a developmental aneurysm, failure of the hypertensive heart, the heart affected by valvular disease, pneumonia and senility account for many deaths. It is interesting to note the close association between a large left ventricle and a terminal cerebral hæmorrhage, also the presence of advanced pulmonary tuberculosis in subjects who have received no medical attention.

An asphyxial mode of death is often seen in young babies. A certain number of these deaths are regularly due each year to suffocation. Many babies are found dead in their cots, frequently face downwards. It is often assumed that these babies have died of suffocation, but many of these deaths are due to natural disease, such as bronchopneumonia, in which the finer ramifications of the bronchial tree fill up rapidly and produce the usual post-mortem appearance of asphyxia. The soft pillow is associated with most of the cases of suffocation. If a pillow must be used, it is better placed beneath the head of the mattress.

The Coroner's Inquest.

A resident medical officer was administering a nitrous oxide and oxygen anæsthetic to a man very ill after appendicectomy. It was thought that the patient had a subphrenic abscess, and during the aspiration of his chest, respiration ceased. The mask was full of semi-fluid material which the patient suddenly coughed up—a liver abscess had burst into the lower lobe of the right lung, and the patient very nearly succumbed to asphyxia. The anæsthetist's greatest fear during that episode was not that the man might die, but the thought that he would have to appear before the coroner as a result of what looked like a certain death on the operating table. This was an unnecessary fear, because in that case probably no inquest would have been held, and even if there had been an inquest, it would have helped to clear the anæsthetist and the surgeon from any suggestion of negligence—an aspect of coroners' inquests too commonly forgotten.

The office of coroner is a very ancient one at common law. Magna Charta (1215) refers to the coroner as "coronator", and the name is probably derived from the title "*custos placetorum coronæ*", or keeper of the king's pleas. A report of an inquest held in 1265 is the oldest report of an English inquest, although there is evidence that coroners existed in the time of King Alfred. In those days a coroner was a very important officer of the crown, and he is still. A text-book written in 1843 states that in his judicial capacity the coroner has the following duties: to inquire—when anyone comes to his death suddenly or violently—how and by what means such death was caused; to pronounce judgements upon outlawries; to inquire of lands and goods and escapes of murderers, treasure trove, wrecks of the sea, fires *et cetera*.

At the coroner's court there is usually no jury. Here the coroner sits to inquire in most cases when, where, how and why a death occurred, and a medical practitioner is generally the most important witness. When there is a jury, it consists of not more than twelve or less than five persons. The inquest is open to the public, although the coroner can if he wishes exclude the public.

In Victoria the *Coroners' Act*, 1938, makes the following provisions:

> Every coroner shall have jurisdiction to hold an inquest concerning the manner of death of any person who is slain or drowned or who dies suddenly or in prison or while detained in any mental hospital within the meaning of the Mental Hygiene Acts and whose body is lying dead within the district in which such coroner has jurisdiction.

In fact the coroner has the power to inquire into any death, natural or otherwise, as he thinks fit. By Section 33, any person, including a doctor, who obstructs a coroner in the performance of any duty under the *Coroners' Act* is liable to a penalty.

The Case to be Reported to the Coroner.

Under the *Registration of Births, Deaths and Marriages Act*, 1938, it is incumbent on the medical practitioner to notify the coroner or the police of certain deaths. In all cases of sudden unexpected death, or when it has come to the knowledge of the doctor that death has occurred from unnatural causes, or under any circumstances of suspicion, or where the medical practitioner is unable to certify the cause of death, the death should be reported. Failure to comply with these provisions involves a penalty of not more than £5.

When a death is reported to the coroner, the local police have instructions to furnish certain information required. This police report is of the utmost value to the pathologist conducting an autopsy—for example, it may contain the only information that the deceased person was an epileptic. Without that knowledge, the pathologist would probably be unable to account for death. The pathologist is not armed with a sheaf of clinical notes and often has no information to guide him in the autopsy. In one case of fatal abortion, it was not disclosed until the inquest that a curette had been performed by the surgeon—a fact that considerably altered the pathologist's interpretation of the presence of a laceration on the cervical wall. When the medical practitioner is reporting a death, he should make a written statement setting out what he knows of the case. If the medical practitioner who attended the deceased person is not prepared to issue a death certificate, his statement should include the following: (*a*) period of attendance; (*b*) date when the subject was last examined; (*c*) particulars of the deceased person's illness; (*d*) whether or not death was expected to be sudden or to occur when it did; (*e*) his opinion of the cause of death. Frequently the coroner, after considering the reports and statements, decides that no post-mortem examination is necessary, that the death was due to natural causes, and accepts what the medical practitioner has stated as the cause of death.

In reporting deaths to the coroner, the following list is a useful guide: (*a*) death by slaying, drowning, injury, accident or violence; (*b*) sudden death in many cases, or when a person is found dead and death is not clearly due to natural causes; (*c*) suicide, homicide, or suspected homicide; (*d*) death of a patient while under an anæsthetic; (*e*) death of an infant aged under five years in a house registered under the *Children's Welfare Act*; (*f*) death of a person detained in a gaol or place of legal confinement, a mental hospital or a private mental home.

The Death Certificate.

The death certificate is bound up with the problem of the case to be reported. Before giving a certificate of death, the practitioner should be satisfied that death has actually occurred. Fraudulent attempts on insurance companies are not unknown. There is no legal obligation on the part of the doctor to view the body after death. This seems to be a weakness in the law. If it was compulsory, it would help to allay that very real fear possessed by some persons of premature burial. Recently one man wrote and suggested that it would be better if the coffin was covered with a blanket and buried under two feet of hay—then an electric bell should be attached to the coffin lid so that if the body was not really dead, attention could be attracted. Another person left a request that her wrists should be cut after death to safeguard against premature burial. However, when practising in the country, I was admonished by a deceased person's executor (the local butcher) for viewing a body after death—the butcher thought that the signs of death were unmistakable to a lay person and that I was after an extra fee.

The doctor should be satisfied as to the cause of death—he must have had an opportunity of making a diagnosis during life. If he has not seen the patient for some time, he should be cautious about giving a certificate. "Whatever a practitioner certifies he should be prepared to affirm on oath." No certificate should be given if death is the result of unnatural causes, however remote in time or

circumstances—for example, fractured neck of the femur in an elderly subject occurring six months before death. In general, certificates should not be issued for subjects who are comatose or moribund when first examined.

In a recent case, a young man when first examined was comatose—his death shortly after was reported to the coroner. At autopsy it was found that the urine gave a heavy red colour with Fehling's solution. It could have been concluded that death was due to diabetic coma, but the analysis of the organs showed barbitone to be present. This required elucidation by the coroner.

It is readily understood that the utmost care and caution should be observed before a medical practitioner signs a cremation certificate. It is too late for exhumation and analysis if subsequent facts or allegations throw doubt on the death certificate.

In one case, in which a man was found dead in bed, autopsy showed that death was due to natural causes. Twelve months later the police began to receive information to the effect that the deceased person's death was due to foul play—allegations not substantiated by the previous autopsy.

In another case relatives, some six months afterwards, questioned whether a wealthy man's death was due to natural causes. The deceased had not been examined by his doctor during the week before his death, and it was alleged that he had suffered during this time from diarrhœa and vomiting, symptoms not consistent with the cause of death as stated on the death certificate. Deceased had been attended by a young widow before his death, and she benefited by the will. One relative was a chemist, another a solicitor. The body was exhumed and there was no trace of poisoning.

There would have been no satisfactory solution in that case had that body been cremated.

The Surgical Death.

Surgical death is perhaps best explained by some examples.

Two motor-cars collided accidentally. An elderly woman passenger in one of them received a blow over the left side of the chest which fractured some of her ribs. She received medical attention, her chest was strapped, and a few days later she died. The doctor in attendance reported the case to the coroner. He was unable to account satisfactorily for her death. Autopsy showed a ruptured spleen with hæmoperitoneum. At the inquest at which the insurance companies were legally represented, some interesting questions were put to the pathologist. Was this condition of ruptured spleen present from the time of the accident? Could it have been diagnosed prior to death? If it had been diagnosed prior to death, need this woman have died?

All these were pertinent questions from the standpoint of insurance and a possible action for damages.

A woman had her gall-bladder removed—she died shortly after the operation from hæmorrhage from the cystic artery. This was disclosed by autopsy as the death was reported to the coroner.

What are the possibilities at an inquest in such a case with regard to the medical practitioner? The coroner could find one of three things: (i) the doctor who attended the patient was criminally negligent in his management of the case—if so, he could be charged with manslaughter; (ii) the doctor was negligent to a degree less than that warranting a finding of manslaughter—a finding, perhaps, of misadventure; (iii) there was no negligence on the part of the doctor—death was accidental.

In the caring for his patient, it is required of the doctor that he render recognized standard treatment. He must give reasonable treatment carefully and conscientiously. He is not expected to be a specialist and practise at specialist standards—errors of judgement honestly made do not constitute criminal negligence.[1] If he has rendered ordinary conscientious treatment, then I do not think that any coroner would bring in a finding other than that the death was accidental.

In the case of the ruptured spleen, it would be difficult for the pathologist to say that the rupture was present from the time of the accident; it would be difficult to prove that there had not been an intrasplenic rupture which later burst the capsule.

[1] The coroner's court is only concerned with criminal, not civil, negligence.

Provided reasonable care and attention are shown by the doctor, then again the coroner's inquest would help to clear the doctor of any imputation of criminal negligence.

There is another interesting aspect of this question. If a patient dies from some injury inflicted by another person accused of a crime, it does not alter the situation in law for the accused if more expert medical attention might have saved the life of the deceased. In law a man is responsible for all the results of his actions, irrespective of whether he has caused the death of another from a primary or a secondary cause. A period of a year and a day is the limit of criminal responsibility. It does not matter whether the victim was suffering from some pathological condition—for example, malarial spleen ruptured by violence—or if the deceased refused medical attention, or died, say, during anæsthesia.

The Medical Death.

Medical death may be exemplified as follows.

A motorist was involved in a collision. He suffered from concussion and was observed for four hours in hospital. No fracture of his skull was found on radiological examination. He was sent home to rest in bed. One week later he left his bed and while walking to the lavatory collapsed and died. Autopsy showed that death was due to coronary atheroma. There was a bruise in the left temporal area of the scalp.

Did the accident one week before have anything to do with that man's death from coronary atheroma? Who can say it was not a factor?

Here is the case of an elderly man suffering from arteriosclerosis in a private hospital, who at night is extremely noisy and disturbs everybody. He is given a quarter of a grain of morphine and one one-hundredth of a grain of hyoscine. This is insufficient to quieten him, and so the dose is repeated. The patient lapses into coma and dies. His death is reported to the coroner; an autopsy is ordered which shows evidence of an asphyxial death consistent with the medication; but severe disease of the kidneys, chronic nephritis and a large carcinoma of one kidney are also found.

Persons with granular kidneys are more susceptible to morphine than others, and when that aspect of the case was discussed with the coroner, no inquest was held, because it was considered that the disease of the kidneys could have been reasonably overlooked and that no negligence could therefore be reasonably imputed to the doctor.

There is one type of medical death of particular interest to the legal profession—sudden death, at work, or on the way to or from work, of the "candidate for death". In cases of sudden death from coronary disease, we often find obstructed, narrowed, thickened, atheromatous and calcified coronary vessels. The youngest subject I have seen was a man, aged twenty years, who dropped dead while working unloading a ship. His left coronary artery was of the type described. Persons with such coronary arteries are likely to die suddenly at any time, usually during or shortly after exertion, but sometimes when in bed asleep, or sitting working at an office desk. In some of these deaths, to the medical mind, the occupation of the deceased person at the time does not appear to have been causative. What the final event is that causes the heart to stop beating is often not determined. Many opinions are held as to the cause, such as ventricular fibrillation, coronary spasm, vagal inhibition, thrombosis in important microscopic vessels *et cetera*; but whatever the cause (and in the present state of our knowledge of pathology this is uncertain), from the medical approach there seem to be no clear-cut cause and effect. The legal approach appears to be different. As was pointed out by P. D. Phillips[(1)] in the evolution of workers' compensation law during this century, the relation between cause and effect was originally very important, but is now less so, because the law is protecting the worker against a risk arising out of being employed, a risk that death might occur incidentally to his employment. The general trend in these medically unsatisfactory cases under workers' compensation law has been to enable the claimant to succeed.

When so many of these persons are found dead in bed, it seems just too bad that the relatives do not benefit by compensation, whereas if the man had lived an hour or two longer to be on his way to work, a claim for compensation would have probably succeeded.

The Problem of the Anæsthetic Death.

There were 23 deaths during anæsthesia last year in Melbourne, and that figure will be approached again this year. It is stated that the anæsthetist and the surgeon are under obligation, by virtue of the *Registration of Births, Deaths and Marriages Act*, 1938, to report such deaths to the coroner—but not under the *Coroners' Act*. I feel that many of these anæsthetic deaths are definitely preventible, and that this is a field for the saving of life, and one deserving of much more attention from the medical profession. Deaths during anæsthesia may result from one or more of the following: (*a*) the disease for which the operation is undertaken—for example, delayed treatment of a ruptured ulcer; (*b*) concomitant disease—for example, coronary obstruction in a patient operated on for inguinal hernia; (*c*) the operation—for example, uncontrollable hæmorrhage; (*d*) the anæsthetic—directly or indirectly. Any autopsy of this nature should be preceded by a thorough discussion between all the parties concerned, because the pathologist is often deprived of many useful data in finding the cause of death. Depending upon the post-mortem findings, the coroner may or may not order an inquest. In the preventible anæsthetic deaths, the result is sometimes due to insufficient attention to the well-established principles of anæsthesia. Asphyxia is the killer. Vomitus inhaled into the air passages during anæsthesia or during the recovery phase is a great danger, as is blood which gains entrance into the bronchial tree during operations about the mouth or throat. After accidents, or if the patient is fearful, the stomach cannot be relied upon to be empty four hours from the previous meal; the emptying time of the stomach may be delayed for hours, and due allowance should be made for this. Artificial respiration may defeat its purpose if the throat is not thoroughly cleared before its institution. Artificial respiration may draw fluid vomitus and blood down into the trachea and bronchi after natural respiration has ceased, and may help to keep material in position that is already well down the bronchial tree.

If a young, healthy subject during ordinary anæsthesia ceases to breathe, great attention should be paid to the respiratory passages. Death in my experience is much more likely in these cases to be due to respiratory obstruction than to sudden cardiac arrest. A tracheotomy and good suction through the tracheotomy wound may occasionally save a life. It is never wise to give an anæsthetic to perform some operation single-handed unless the exigencies of the situation compel it. There is no such thing as a minor operation under general anæsthesia. Any anæsthetic carries a risk and should be given preferably under ideal surroundings.

At an inquest on such a death, the coroner wants answers to the following questions: (*a*) What was the cause of death? (*b*) Was an anæsthetic necessary? (*c*) If so, was the most suitable anæsthetic agent chosen? (*d*) Was it of standard purity? (*e*) Was it administered with reasonable care and skill? (*f*) When untoward symptoms manifested themselves, what resuscitative steps were taken to overcome them? (*g*) Was the deceased carefully examined before anæsthesia to detect signs and symptoms of disease? (*h*) If the operation was in progress, was surgery proceeding according to the recognized standards?

The anæsthetist has little to fear and much to gain at any inquest if he has shown the ordinary skill and care required of him.

The Problem of the Alcoholic.

The problem of the alcoholic is a subject of great interest to the pathologist as well as to the medical practitioner. Valuable evidence is often missed because a sample of blood is not taken after death and its alcohol content estimated. Such a procedure sometimes helps to determine the cause of death. This should be done in all cases of crime—in all major cases (murder and manslaughter). When blood is taken after death, it should be taken from a peripheral vein and nowhere else if it is proposed to use the quantitative alcohol level as evidence. The test is not reliable if the blood is taken elsewhere, except qualitatively; it is not reliable if the blood is taken from the heart unless very close to death; and it is by no means reliable if the blood is picked up from the body cavity during post-mortem procedure.

Within the first three days after death, the alcohol level of blood from a peripheral vein is a reliable index of the blood alcohol level at the time of death. Alcohol present in the stomach at death may make its way through the stomach wall, the œsophagus and the intestine after death, and it may diffuse along veins. It is for that reason that blood from a peripheral vein is required for the test. Absence of alcohol or its odour from the stomach is compatible with a fairly high blood alcohol level. The level at death is not altered by the production of volatile reducing substances due to post-mortem changes.

A man fell from a motor bus, the wheel of which passed over his head. Twelve hours after death, his blood alcohol content was 410 milligrammes *per centum*. In the face of this finding, it would be unlikely that any action for damages against the bus driver would be successful.

An unknown woman was found floating in the Yarra. She had been dead about three days. Her blood alcohol level was 390 milligrammes *per centum*—a finding that would allow the reasonable inference that she fell into the river whilst considerably under the influence of alcohol.

A post-mortem investigation of the blood alcohol content often throws much light on the cause of a fatal road accident, when either a pedestrian or a motorist is involved. One case calling for care to avoid a miscarriage of justice was that of a motor truck driver who had been driving very erratically before he finally drove into an oncoming vehicle and was killed. His erratic driving was accounted for on the basis of a much abused term, "blackouts", whereas I thought it was due to alcohol—and that was what stimulated some research on the question of the post-mortem blood alcohol content. If this was estimated as a routine procedure after fatal road accidents, I am sure the results would be most enlightening.

Reference.

(1) P. D. Phillips: "Proceedings of the Medico-Legal Society of Victoria", Volume IV.

OCCULT BLOOD IN FÆCES: THE BENZIDINE TEST.

By HOWARD W. LINN,
Adelaide.

WHEN a patient is receiving iron by mouth as treatment for anæmia due to gastro-intestinal hæmorrhage, it is often important to find out whether blood is still being lost. The stools will be black from the ingested iron, and so an occult blood test must be performed.

Kolmer[1] states that "since the benzidine and other tests are extremely sensitive, the patient should be on a meat-free diet for at least three days before a positive reaction is regarded as significant" and also that "this properly includes abstinence from fish, as well as from broths and soups made from meat stock". Harrison[2] and Hawke and Bergeim[3] agree with these statements.

Most people refrain from giving iron in any form during the test. In view of this a series of experiments was arranged. The results of this investigation are summarized in Table I.

The patients used in these experiments had no abdominal symptoms at all and were mostly suffering from fractures; patients suffering from hæmorrhage from the gums, nose and hæmorrhoids were excluded.

Ferri Carbonas Saccharatus, Ferri et Ammonii Citras, ferrous sulphate and blood were tested directly on several occasions by the benzidine test. All the iron compounds

TABLE I.

Group.	Diet.	Iron *et cetera*.	Results.
Control I	Meat free three days.	——	21 patients tested; one positive result.[1]
Control II	High meat content.	——	20 patients tested, 5 positive results.
Experiment I	Meat free three days.	*Ferri Carbonas Saccharatus*, 20 grains three times a day.	5 patients tested; all results negative.
Experiment II	Meat free three days.	*Mistura Ferri et Ammonii Citratis*, half an ounce three times a day.	5 patients tested; all results negative.
Experiment III	Meat free three days.	Ferrous sulphate, 5 grains three times a day.	5 patients tested; all results negative.
Experiment IV	Meat free three days.	Ferrous sulphate 5 grains three times a day, also meat extracts, for example gravy, "Bovril" and soups.	10 patients tested; all results negative.

[1] In the "positive" specimen muscle fibres were found microscopically, and the patient admitted eating a meat sandwich on the second day of the test.

gave repeatedly negative results. With blood, however, the results were different; it was found that less than 0·01 millilitre of blood gave a positive reaction.

The method used for testing is as follows. A portion of fæces, the size of a marble, is emulsified in ten millilitres of water and boiled for a few minutes. This emulsion is filtered drop by drop into a test tube containing equal parts of hydrogen peroxide and a freshly prepared saturated solution of benzidine in glacial acetic acid. A deep blue colour, developing within a few minutes, indicates the presence of blood.

In somewhat similar experiments on eighteen patients Schwartz and Vil[4] found that stools ceased to react to the benzidine test after three days of meat-free diets, and that iron preparations in most cases did not influence the test.

Iron compounds, given by me in 25 cases, failed to produce any positive results, and so withholding of these is unnecessary.

The most surprising of our findings was that meat extracts, such as gravy, "Bovril" and soups, did not influence the result of the test in any of ten cases. Although the number of cases is small, the fact that no positive results were obtained is of some significance.

Summary.

1. A series of experiments was done with the benzidine occult blood test. The method used gave positive results with less than 0·01 millilitre of blood.

2. A meat-free diet for three days is all the preparation required for the test.

3. Iron compounds and meat extracts did not influence the outcome of the test.

Acknowledgement.

I wish to thank the medical superintendent of the Royal Adelaide Hospital for permission to publish this paper.

References.

(1) J. A. Kolmer: "Clinical Diagnosis by Laboratory Examinations", First Edition Revised, 1944, pages 272 and 1059.
(2) G. A. Harrison: "Chemical Methods in Clinical Medicine", Third Edition, 1947, page 498.
(3) P. B. Hawke and O. Bergeim: "Practical Physiological Chemistry", Eleventh Edition, 1938, page 372.
(4) S. O. Schwartz and C. S. Vil: "Benzidine Negative Stools during Iron Therapy", *The Journal of Laboratory and Clinical Medicine*, Volume XXXII, 1947, page 81.

Reviews.

AN INDEX OF TREATMENT.

"AN INDEX OF TREATMENT", a comprehensive volume by various writers, and edited by Sir Robert Hutchison, assisted by Reginald Hilton, is in the thirteenth edition; it has been delayed for some years because of World War II.[1] The contributors have had a race against time in their attempt to bring everything up to date, and at the same time to maintain the book's range of information. Such a book intended for immediate reference is invaluable to the medical practitioner in the sparsely populated area, remote from consultants, but is also indispensable to the general practitioner in the larger centre as a check upon his thoroughness in diagnosis and treatment. The obligation upon the editors and publishers is therefore all the greater to be sure that each successive edition brings before the reader the most efficacious as well as the most recent medicaments and procedures available. This edition does not fall short in this respect.

The advantage of this particular index lies in the fact that the editors have, without being discursive, ensured ample description of the symptoms and signs of the diseases and emergencies to guide the practitioner in planning his line of treatment. The introduction, written by Sir Robert Hutchison himself upon the general principles of therapeutics, could be read by the most experienced practitioner as a standard for comparison with his own methods of approach, but it certainly should be studied carefully by every novice in the practice of medicine. It combines breadth of view with the scientific outlook, and reminds us that we are dealing with sick human beings as well as applying our professional knowledge of therapeutics to recognized pathological conditions. This work does not pretend to give all the modes of treatment available in any illness, nor does it touch upon labour either complicated or otherwise; but those responsible are to be congratulated upon the vast fund of useful knowledge between its covers. In such emergencies as intestinal obstruction, the general lines to be followed are pointed out; the article upon acute osteomyelitis is concise and complete; fractures of the long bones are included, and the necessary general lines of treatment are outlined before each particular anatomical structure is dealt with. Such articles provide a valuable aid to those required to deal with emergencies in these times of shortage of hospital beds. The subject of the care of the dying is too often either ignored or brushed aside by those who teach the medical student and the hospital resident medical officer, but an excellent article is devoted to this important responsibility of the profession; it should be studied by every graduate in medicine, and if properly applied would add to the honour of the medical profession both in private and in hospital practice. Because of its comprehensiveness, its practical outlook and its conciseness, this book should be at hand for every medical practitioner, especially in his early years of service to his fellow men.

[1] "An Index of Treatment", edited by Sir Robert Hutchison, Bt., M.D., LL.D., F.R.C.P., assisted by Reginald Hilton, M.A., M.D., F.R.C.P., and contributors; Thirteenth Edition; 1948. Bristol: John Wright and Sons, Limited. 9¼" × 6¼", pp. 984, with illustrations. Price: 84s.

HUMAN NEUROANATOMY.

THE second edition of Strong and Elwyn's "Neuroanatomy" has just appeared (1948).[1] On the whole it is a well-balanced text-book, incorporating or referring to much of the recent work in both neuroanatomy and neurophysiology, and includes a useful bibliography for reference purposes. The parts upon the blood supply, meninges and cerebrospinal fluid pathways are excellent; but it is regrettable that few American authors make acknowledgement of the excellent work of Abbie upon the forebrain arteries, which preceded that of Alexander by three or four years, while in the chapter on the meninges the illustration of the cisterns could well be improved as we have nowadays such excellent figures as those of Max Brodel or Mettler.

Chapter II on the morphology of development contains a useful summary, incorporating figures from the more

[1] "Human Neuroanatomy", by Oliver S. Strong and Adolph Elwyn; Second Edition; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 6¼", pp. 456, with illustrations. Price: 45s.

recent work of Hochstetter, while the chapters on the neuron, neuroglia and histogenesis are excellent; the figures illustrating cross-sections of the *medulla oblongata*, pons and midbrain are also good, and compare favourably with those of other similar text-books. In general criticism, some minor defects are noted, namely, that the autonomic nervous system could be more fully treated, especially with regard to visceral afferent pathways, that recent work on the dermatomes, so important in intervertebral disk and spinal cord work, has not been incorporated, that tract overlap in the spinal cord might receive a little more emphasis, and that the treatment of the thalamic nuclei and their connexions, and especially the optic pathway, could be improved. Also no mention is made of recent work on the relation of size to function in myelinated nerves, while the controversial statement is made that muscle spindles are especially abundant in the small muscles of the eyes (page 75). The cerebellum, especially its histology, is beautifully illustrated, while the chapters on the hypothalamus and cortex are excellent, the latter incorporating Lorente de No's and Bucy's recent work. In conclusion then, this work can be recommended to the medical student, or to the graduate studying for a higher degree, as a suitable alternative to other well-known works on neuroanatomy, although in our opinion those features indicated above need further elaboration.

SURGERY OF THE COLON AND RECTUM.

IN the introduction to "The Surgery of the Colon and Rectum" by Sir Hugh Devine and John Devine, it is stated that the book may appear to have a personal touch.[1] The book has a pronounced personal and family touch and thus has unusual value. Surgeons may disagree with some of the surgical procedures advocated in the pages of this book, but they will readily admit that the senior member of the trio has for the past quarter of a century held his position as one of the leaders in abdominal surgery in Australia.

The work covers a wide field and describes the anatomy, the comparative anatomy and the physiology of the colon and rectum, and also their affections—both simple and malignant—with appropriate measures for relief. There are thirty-two chapters making up the three hundred and sixty-two pages including two hundred and seventy-seven drawings and photographs, many of which are coloured. The index is detailed fully, and throughout the pages, when statements are made, illustrations are handy to indicate the intentions of the authors. The type is easy to read, the paper is of nice texture and Miss Devine's drawings show considerable talent. The drawings are much more explicit than the photographs.

In an Australian treatise on such a subject, reference to the work on rectal supports by that distinguished Australian, Elliott Smith, was expected, but was sought for in vain. Looking at Figure VII, the student will be perplexed as to what blood vessels nourish the rectum.

The importance of an accurately taken history as the principal factor in the making of a correct diagnosis is stressed.

It would be fitting to find some tribute to the monumental work of Kraske and Miles in developing the surgery for cancer of the rectum, but none appears. Kraske sixty years ago warned surgeons against working above the third sacral nerve and stressed the danger of injuring it. Mention of this is made, but not of Kraske, who even defunctioned the distal part of the colon and rectum by occasionally using a right-sided colostomy.

One chapter is given up to the description and use of the Devine frame. Modern anæsthesia gives such excellent muscular relaxation that, today, retractors other than the help of the delicate touch of the expert assistant are frequently dispensed with.

The mobile transverse colon with its attached omentum can be a great friend to fill the gap caused by removing the whole or part of the colonic canal from the splenic flexure to the rectum, but not if it is fastened to the upper part of the abdomen by being pulled out to make a colostomy as a preliminary to resection lower down.

It is felt that perhaps too much importance is given to the defunctioning of the colon at its transverse section, and some will question the wisdom of cutting the colon and bringing out the two cut ends through separate button-hole incisions in the skin.

Exploration of the abdomen in cases of obstruction through a two-inch incision in the upper right rectus muscle is recommended. Surgeons who wear a seven-sized glove would find difficulty in getting their hand in through such a small incision, and even if they did so would be afraid of inflicting trauma on the intestines, especially in cases of obstruction with distension. The old surgical adage "when in doubt in cases of intestinal obstruction explore in the middle line below the umbilicus" has still much to recommend it.

The modern use of X rays to define bowel distension and the use of an opaque enema, clysma or clyster—whatever term is fancied—as advised many years ago by H. S. Newland, will locate the site of obstruction.

Why dispense then with the Paul-Mikulicz operation for left-sided colonic growths which enables the surgeon at one fell swoop to relieve obstruction and remove the growth? In cases of acute colonic obstruction, since fully 95% of colonic cancers are distal to the ascending colon, no surgical procedure can be more life-saving than a preliminary muscle-splitting incision over the caecum and, if distended, caecostomy; yet the operation is just casually mentioned.

Figure 235 shows an array of stations for two teams of surgeons, assistants and apparatus to excise a rectum and is awe inspiring, while Figure 236 shows the surgeon carrying his instruments—including sharp scalpels—in his gown and is indeed a novelty.

The modern tendency in the treatment of rectal cancer to revive the long ago discarded operations for preserving the anus is discussed and an operation is shown in Figure 212 which is a replica of that described by Donald Balfour in 1910. The value of any operation anywhere in the body for eradication of cancer can be assessed only by the recurrence rate. This should be kept in mind when conservative operations on the rectum for cancer are contemplated. The perineo-abdominal operation, which is strongly advocated, has little to recommend it once the Miles technique has been mastered, but it has to be admitted that this is not easy to do.

To defunction the transverse colon before excision of a rectum is a debatable procedure, adding further discomfort to the unfortunate victim, unless the aim is the restoration of the continuity of the bowel and preservation of the anus; but, when a permanent left inguinal colostomy is the ultimate aim such an operation has no place.

With the advent of the Miller-Abbott tube, the "sulpha" drugs, blood transfusion and administration of appropriate vitamins, the performance of a colostomy by use of the transverse colon as a preliminary to operation on the colon beyond and on the rectum, will tend to recede into the background.

This work has considerable merit, is full of original thought and is a credit to both authors.

[1] "The Surgery of the Colon and Rectum", by Sir Hugh Devine, M.S. (Melbourne), Hon. F.R.C.S. (England), F.R.A.C.S., F.A.C.S., and John Devine, M.S. (Melbourne), F.R.C.S. (England), F.R.A.C.S., F.A.C.S.; 1948. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall (1941), Limited. 8½" × 5½", pp. 374, with many illustrations, some of them coloured. Price: 52s. 6d.

MIDWIFERY FOR NURSES.

THE very fact of the appearance of a fourteenth edition vouches for the merit of Jellett's "Short Practice of Midwifery for Nurses".[1] In this latest revision by Dawson the general character of the book is preserved, while new material has been added, notably in the consideration of puerperal infection, the use of radiology in obstetrics, and hæmolytic disease of the newborn.

From the opening chapter the impression is gained of a determination to instil into the minds of pupil midwives that much can be done to avoid obstetric pitfalls by careful attention to detail and by intelligent observation. Where timely medical aid cannot be summoned, action to be taken in the event of emergencies such as ante-partum and post-partum hæmorrhage is described in detail.

There is a departure from conventional usage in respect of the terms "miscarriage" and "abortion", these being described as separate entities on the basis of the period of gestation involved. The use of hot vaginal douches as a treatment for primary uterine inertia is not regarded as good treatment. In severe preeclampsia and eclampsia

[1] "A Short Practice of Midwifery for Nurses", by Henry Jellett, B.A., M.D. (Dublin University), F.R.C.P.I., and J. Bernard Dawson, K.B.E., M.D., B.S. (London), M.D. (Adelaide), F.R.C.S. (England), F.R.C.O.G., F.R.A.C.S.; Fourteenth Edition; 1948. London: J. and A. Churchill, Limited. 7" × 4½", pp. 474, with illustrations. Price: 12s. 6d.

treatment includes gastric and bowel lavage—a form of therapy no longer widely practised; but as the details of management of these conditions are included for the information only of the nurse, perhaps this is but a minor criticism.

This useful volume fulfils its purpose of presenting to the pupil midwife the essentials of her art, and a thorough understanding of its teachings will enable her to carry out her duties with confidence and success.

FEVERS

BREEN'S "Essentials of Fevers" contains within a relatively small space the essential information on infectious diseases for students and practitioners, for whom it is primarily intended.[1] The latest edition has been enlarged by the addition of colour plates which, though diagrammatic, are more convincing than the poorly reproduced photographs so often seen. There are some interesting "notes" on sore throat and food poisoning, as well as new sections dealing with typhus, glandular fever, undulant fever, influenza and *encephalitis lethargica*. These additions have been made without great interference with the size and convenience of the work. The book thoroughly deserves the popularity it has achieved in Britain, and merits a wider reading public here.

Notes on Books, Current Journals and New Appliances.

PSYCHIATRIC EXAMINATION CARDS.

A SECOND edition has just been issued of the psychiatric examination cards devised by A. Spencer Paterson which were first published in 1943.[2] They are pocket size and sold in a transparent envelope. On one side are the questions asked at the first interview with a psychiatric patient in order to establish a provisional diagnosis. On the other side are questions put to a patient with psychoneurosis to elicit causal factors in the history. The cards are intended for use not only by psychiatrists, but by all medical practitioners who encounter psychiatric patients in their practice.

AN INDEX OF MODERN REMEDIES.

MOST practitioners find it difficult to keep up with the ever-growing list of modern pharmaceutical preparations and practically impossible to know the multitude of their names, proprietary and otherwise. "An Index of Modern Remedies", by William Mair,[3] will be found useful in coping with this situation. The latest edition of this publication, which is stated to be for the medical and pharmaceutical professions only, is in line with the British Pharmacopœia, 1948, and contains a summary of the principal changes (additions, deletions *et cetera*) of the new pharmacopœia; the newly admitted preparations are listed with the names of their proprietary forms, their chemical constitution and their therapeutic application. A list of the proprietary names of many British, American and Continental preparations is given with their respective chemical constitutions. Then follows an extensive survey of modern remedies, grouped mainly according to their therapeutic use, with details of manufacturers, packing and price. A key to the names and addresses of manufacturing chemists is also included. Finally there is a detailed index which should lead the searcher to the information needed about any particular preparation. This little publication is unpretentious in its make-up, but it has obviously required much work in preparation and will save a great deal of work and confusion for those who use it. It is to be commended.

[1] "Essentials of Fevers", by Gerald E. Breen, M.D., B.Ch. (N.U.I. Dub.), D.P.H., D.O.M.S. (R.C.P. London, R.C.S. England); Second Edition; 1948. Edinburgh: E. and S. Livingstone, Limited. 7¼" × 4½", pp. 366, with many illustrations, most of them coloured. Price: 15s.

[2] "Psychiatric Examination Card: Extended History in Cases of Neurosis", by A. Spencer Paterson; Second Edition; 1948. London: H. K. Lewis and Company, Limited. 5½" × 4¼". Price: 9d. net.

[3] "An Index of Modern Remedies", by William Mair, F.R.S.E., F.C.S., M.P.S.; Fourth Series; 1948. Glasgow: The Scottish Chemist. 9½" × 7". Price: 3s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Diabetic A B C: A Practical Book for Patients and Nurses", by R. D. Lawrence, M.A., M.D., F.R.C.P. (London); Tenth Edition; 1948. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 92. Price: 4s.

A practical handbook for patients, written by a diabetic for diabetics.

"Liver Injury"; Transactions of the Seventh Conference, January 15 and 16, 1948, New York, N.Y.; edited by F. W. Hoffbauer, M.D.; 1948. New York: Josiah Macy, Junior, Foundation. 9½" × 6½", pp. 101.

A verbatim report of the conference, the fourth in the series.

"Critical Studies in Neurology", by F. M. R. Walshe, M.D., F.R.S.; 1948. Edinburgh: E. and S. Livingstone, Limited. 8½" × 5½", pp. 276, with illustrations. Price: 15s.

A series of studies reprinted from *Brain* and from the *British Medical Journal*.

"The Practice of Endocrinology", edited by Raymond Greene, M.A., D.M., M.R.C.P.; 1948. London: Eyre and Spottiswoode (Publishers), Limited. 9½" × 6", pp. 392, with illustrations, some of them coloured. Price: 52s. 6d.

Intended for the general practitioner, stress being laid on practice rather than on theory.

"Obstetrics and Gynæcology: A Synoptic Guide to Treatment", by Beatrice M. Willmott Dobbie, M.A., M.B., F.R.C.S., D.M.R.E.; 1948. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 376, with illustrations. Price: 20s.

Intended chiefly for the general practitioner, but also for candidates for higher examinations.

"Eden and Holland's Manual of Obstetrics", by Alan Brews, M.D., M.S. (London), M.R.C.P. (London), F.R.C.S. (England), F.R.C.O.G.; Ninth Edition; 1948. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 814, with illustrations. Price: 42s.

Reflects the teaching and practice of obstetrics at the London Hospital.

"Clinical Endocrinology: For Practitioners and Students", by Laurence Martin, M.D. (Camb.), F.R.C.P. (London), and Martin Hynes, M.D. (Camb.), M.R.C.P. (London), foreword by Sir Lionel Whitby, C.V.O., M.C., M.D., F.R.C.P., D.P.H.; 1948. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 236, with illustrations. Price: 15s.

An account of endocrine disease and of hormone therapy written from the point of view of the general physician.

"A Method of Anatomy: Descriptive and Deductive", by J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edin.); Fourth Edition; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 8", pp. 884, with many illustrations. Price: 56s.

Intended to be "a working instrument designed to make anatomy rational, interesting and of direct application to the problems of medicine and surgery".

"Fractures and Dislocations for Practitioners", by E. O. Geckeler, M.D.; Fourth Edition; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 5¾", pp. 388, with many illustrations. Price: 37s. 6d.

A summary of the subject intended as a complete, yet simplified, guide to treatment.

"The Medical Annual: A Year Book of Treatment and Practitioner's Index", edited by Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P., and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; 1948. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall (1941), Limited. 8" × 5¾", pp. 556, with illustrations, some of them coloured.

The sixty-sixth consecutive volume bearing this title.

"Clinical Roentgenology of the Digestive Tract", by Maurice Feldman, M.D.; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 5¾", pp. 916, with 641 illustrations. Price: 60s.

Written as an aid to diagnosis of digestive tract conditions for the use of radiologist, gastro-enterologist, student and general practitioner.

"Exercise and Physical Fitness: An Introduction to the Study of the Theory and Practice of Bodily Exercise", by George Zephirin Dupain; 1948. London, New York, Sydney and Paris: Shakespeare Head Press Proprietary, Limited. 8½" × 5½", pp. 270, with illustrations. Price: 20s.

The author's aim is to contribute elementary material for the lay public as well as for teacher and student and to correlate the theory of exercise with its practice.

The Medical Journal of Australia

SATURDAY, JANUARY 1, 1949.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE AUSTRALIAN POST-GRADUATE FEDERATION IN MEDICINE.

A few years ago it was suggested in these columns in a discussion on post-graduate medical training that the term student should not be reserved for undergraduates at medical schools and that a healthier idea of values would be shown if medical graduates were classified as medical students and medical drones. Since that time an increasing number of medical graduates have learned that private study of journals and books, although indispensable, is not enough. That they have come to this conclusion is clear from the large numbers who attend the post-graduate courses of study that are organized in different parts of Australia. The history of post-graduate teaching in the Commonwealth is full of interest and reflects the constantly increasing needs of the practising members of the profession. Medicine has in the last fifty years made constant progress, but latterly there has been an acceleration in the advance and there is no indication that a slowing down is likely to take place. This means that post-graduate medical education will become even more necessary in the years to come than it is today. The development of post-graduate teaching in Australia has been followed by this journal with critical interest ever since the Melbourne Permanent Post-Graduate Committee was brought into being by the Victorian Branch of the British Medical Association. It was not long after this that post-graduate committees were established in other States, and it soon became apparent that if the several committees were to be really useful to the practitioners of the Commonwealth they would need to conduct their activities in close liaison with one another. It was in April, 1934, that a plea was made in these columns for the establishment of a Federal Council for Post-Graduate Work. On that occasion it was pointed out that the infrequent courses of instruction held at that time sometimes overlapped and also that when the visits of overseas lecturers were contemplated there should be some means of pooling resources so that one lecturer might visit each centre in turn. In 1934 we remarked that rumours had been heard that something was likely to be done, and since then discussions for the federation of post-graduate committees have actually been held. For one reason or another agreement was not reached, much to the disappointment of everyone concerned. At long last, however, the formation of the Australian Post-Graduate Federation in Medicine has been announced. This is a matter for congratulation and an occasion on which we may justifiably indulge in high hopes for the future.

The objects of the new body are stated in its constitution to be "the encouragement and advancement of post-graduate medical education, work and research". The members of the Federation are set out in two groups. The first includes the following post-graduate committees: The Melbourne Permanent Post-Graduate Committee, The Post-Graduate Committee in Medicine in the University of Sydney, The Post-Graduate Committee in Medicine of the University of Adelaide, The Post-Graduate Medical Education Committee of the University of Queensland, The Western Australian Post-Graduate Committee (British Medical Association), The Tasmanian Post-Graduate Committee in Medicine. The second group comprises "such other organizations (including any New Zealand or other overseas organization) as the Federation may admit to membership from time to time upon such terms and conditions as it may prescribe". The inclusion of this second group has been wise and it would be safe to predict that before long "other organizations" will be admitted to membership. The Federation will be controlled by its "members" and it is to be noted that at least two general meetings are to be held in each year—one an annual general meeting during the latter half of the calendar year, and at least one other general meeting at such time and place as the Executive Committee, with the approval of the President, shall determine. Between general meetings the affairs of the Federation will be controlled by an Executive Committee. This Executive Committee is to be elected from persons "nominated by the Post-Graduate Committee of the State chosen for this purpose". The Post-Graduate Committee in Medicine in the University of Sydney was selected to nominate the Executive Committee for the first year. The Executive Committee has been elected and office-bearers have been appointed for the year commencing October 20, 1948. Colonel A. M. McIntosh is the first President and Dr. S. A. Smith the first Vice-President. The members of the Executive Committee are Professor E. Ford and Dr. R. H. Macdonald, with Dr. George Bell as Honorary Treasurer and Dr. Victor M. Coppleson as Honorary Secretary. The Federation is to be financed by the contributions of the members and these have been arranged so that the greatest contributions are to be made by the largest States. Thus New South Wales will pay eight parts, Victoria eight parts, Queensland and South Australia each four parts, Western Australia two parts and Tasmania one part. It is understood that the formation of the Federation will in no way limit a committee in the exercise of its own independent functions in its own State; but what these independent functions are is not stated in the constitution of the Federation.

The new Federation is clearly constituted in such a way that it should function without any hitch or even difficulty. In all matters covering the whole of the Australian States long distances, climatic conditions and custom help to alter the individual practitioner's point of view and make it easy for misunderstandings and actual differences to take place. In this instance the objects of the Federation are so general and the ground to be covered is so wide

that serious differences of opinion should not occur; and, since local initiative on State functions has been preserved, the possible reasons for their occurrence have been made fewer. There are two spheres in which the Federation can be immediately effective—the visits of overseas lecturers to Australia and the studies of Australian graduates in overseas centres. When medical teachers and lecturers from the other side of the world visit Australia the arrangements cannot be said to be complete unless medical graduates in all the States have an opportunity of hearing the lecturer. To bring a teacher of renown to this part of the world from the centres of learning in Britain or America costs money—the travelling expenses have to be met and a sustenance allowance has to be provided. In addition, as is fitting and proper, a honorarium is as a rule paid to the lecturer. A federation of post-graduate bodies can more easily finance such a venture than one body acting alone, or even two or three acting together. The past has shown that visits from overseas medical lecturers are appreciated in every part of Australia, and incidentally we may suspect that the lecturers gain from their journeyings both pleasure and experience which is of value to them. What will probably happen is that names will be suggested by the several members of the Federation, that a selection of these will be made in order of preference and invitations then be issued. We can imagine that occasions will arise on which invited lecturers will find it hard to arrange their affairs so that they will be able to accept, and in those circumstances a second string to the bow is necessary. We understand that steps are already being taken to select a lecturer for the latter half of 1949. In regard to the studies of Australian graduates in overseas centres the existence of the Federation will make the liaison between Great Britain and ourselves closer than it is at present. The British Postgraduate Medical Federation, of which Sir Francis Frazer is the director, will find it much more satisfactory to deal with one organization in this country than with six separate bodies. In the past some graduates desirous of studying in Britain have gone to England and have applied in London to the British Postgraduate Medical Federation without any recommendation from an Australian body. Sometimes they have written to England announcing their intended arrival and the London Federation has not been able to do what it wished to do because there was no single body from which it could make inquiries. Sir Francis Frazer and his fellow officers wish to do away with the idle period experienced by casual arrivals looking for post-graduate instruction and resident appointments. We look forward to the establishment before very long of arrangements which will be completely satisfactory. But one further statement remains to be made—It is not only with Britain that the Australian Post-Graduate Federation in Medicine will be able to deal, nor even with the United States in addition, but with other units of the British Commonwealth of Nations. We look forward to the time when Australian graduates will go to study in these places and when students from all the units of the Commonwealth will come to Australia. This is, of course, a long-range policy and it is a far-flung hope. That it will happen is certain if everyone concerned wills it to happen. The Australian Post-Graduate Federation in Medicine will probably have some share in such a happening, but that is a subject for a future discussion.

Current Comment.

CHILDBEARING AFTER THE AGE OF FORTY YEARS.

WHEN a woman over the age of forty years asks whether it is wise for her to have a baby the answer is not always easy to give, whether she has already borne children or not. Her desire for the child may be great and she may well bear it successfully, but most practitioners feel uneasy about the matter; many will have had only limited experience of such births and little has been written about them. For this reason a study by M. E. Davis and A. Seski[1] of the records of 1011 women, aged forty years and older, delivered at the Chicago Lying-in Hospital, is welcomed. These 1011 deliveries were consecutive, occurring during the years 1927 to 1944. During the same period a total of over 52,000 women were delivered at the hospital. Of this large number only two were aged forty-six years and two were aged forty-eight years, facts confirmatory of the rarity of childbirth in women over the age of forty-five years. The first finding of importance was that the complications of pregnancy were much more common in this special group of patients than in the general population of the hospital. One out of every four women developed toxæmia of pregnancy. *Placenta prævia* and *abruptio placentæ* occurred much more often than in the general hospital population. As might be expected, circulatory complications other than the toxæmias (including heart disease, thrombophlebitis, phlebothrombosis, and extensive varicosities), which are much commoner in middle age than in the young, increased the hazard of childbearing. Labour terminated uneventfully in the majority of cases, but there was a great increase in the incidence of operative intervention as a result of the complications of pregnancy and the age of the members of the group. There were no maternal deaths that could not be prevented by modern obstetrical methods. Turning from mother to infant, we find that babies born to women aged forty years and older shared in the greatly increased risks of childbearing. One out of every ten mothers failed to take a baby home with her when she left hospital—a dramatic statement, no doubt, but a highly significant one for this group of women. The high fœtal mortality was the result of an increased number of premature babies, an increased number of congenital abnormalities, and a higher incidence of fœtal damage because of the greatly increased number of pregnancy complications. It is also important to note the relative frequency of fœtal abnormalities not necessarily fatal, such as mongolism. "The increased hazard of a congenital anomaly in the baby cannot be dismissed lightly in women of 40 and older"—again a significant statement for these women, the majority of whom want a baby very much. Concerning the post-delivery period, Davis and Seski were unable to find out a great deal; they comment that it has been their experience that elderly women recover more slowly than younger women, but also draw attention to the remarkable rejuvenation that may occur in these mothers after childbirth. The subsequent story is not entirely relevant to the present study, but physical and psychological problems do arise for the middle-aged mother, especially of an only child, which are unknown to younger women, and they, too, should be known to the prospective mother. As a final thought Davis and Seski raise the question of the amount of permanent damage that may have followed pregnancy complications and the possibility of a resultant handicapping or shortening of the mother's life. These are questions not yet fully answered and they warrant more study. They are most important, particularly for a multiparous woman with children who need their mother to help them to grow up normally and happily. Even, however, without the answers to these questions, this Chicago study is certainly helpful, and supplies a background of facts. The risks are very real, but with sound medical care many pregnancies reach a thoroughly satisfactory conclusion. It is

[1] *Surgery, Gynecology and Obstetrics*, August, 1948.

right that parents should know the hazards faced by a woman over the age of forty years who would have a baby, so that they might be spared not only false hope and bitter disappointment, but also vague and unwarranted fears.

CAPILLARY LESIONS IN ARTERIAL HYPERTENSION.

THE hæmorrhagic lesions occurring in the course of arterial hypertension have often suggested to clinicians the possibility of foretelling such threats to a patient's safety. The importance of the capillaries not only in maintaining the circulation efficiently but also in holding out against the stealthy inroads of age has been recognized, although measure of their functional capacity is possible to only a limited extent. The "capillary fragility test" is one of the efforts to look into the future of the hypertensive patient and to attempt a forecast of the degree of danger which may threaten his vascular system in a vital place. Louis A. Soloff and C. T. Bello have investigated this problem from two points of view, the validity of the capillary fragility test, and the connexion if any between the occurrence of retinal hæmorrhages and cerebral vascular lesions.[1] Reports have been made of series of tests on hypertensives which seemed to show a large proportion of reactors, but other work has not confirmed this. One reason for this is apparently the lack of agreement as to the technique of the test. When the cuff of a sphygmomanometer is applied to the arm petechiæ may be produced below the cuff under certain conditions of pressure, and as might be expected, they appear more readily as the amount and duration of the pressure increase. The question at issue in Soloff and Bello's work is whether the pressure is above or below the diastolic level individual to the subject of the test. They also point out that subclinical scurvy has been shown to be not uncommon, and will vitiate the test for this particular purpose. Therefore in investigating a series of 50 hypertensive patients they saturated them with vitamin *C* for a month. Though 33 patients showed a positive result with the test when the supra-diastolic pressure was used, only two responded to the test when the infra-diastolic pressure was used. The effect of rutin was tried on the last-mentioned two patients, but no reversal of the result of the test could thereby be obtained. Further, on comparison of the patients who showed petechiæ in the test with those in whom retinal hæmorrhages had been observed, no correlation could be found.

The relationship of retinal bleeding to cerebral hæmorrhage if any was now investigated. The records of hospital patients over a five-year period were examined, and those were selected for study which dealt with subjects of an established hypertension who had died or who had suffered a cerebral vascular accident, and who in addition had been observed to have retinal hæmorrhages by chosen experts in ophthalmology. The figures were submitted to analysis, and although they were too small to be unassailable from the statistical point of view, the conclusions drawn appear to be justified. It seems to be clear that retinal hæmorrhages cannot be regarded as prognostic guides as to the likelihood of cerebral hæmorrhage. The reverse is rather true according to Soloff and Bello, who found that a hypertensive patient with retinal hæmorrhages was more likely to die from cardiac or renal failure. In this study patients with retinal hæmorrhages were more commonly found among the group suffering from uræmia of renal or extrarenal origin than any other. Analysis of the types of hypertension apparently prone to produce retinal lesions indicated the importance of spasm: a high degree of spasm and a well-advanced degree of sclerosis always accompanied them. The authors suggest that the occurrence of arteriolar spasm increases the burden on the heart and kidneys, and that the patient is then in danger of not living long enough to suffer a cerebral accident. The actual figures found for patients with retinal hæmorrhages who had cardiac failure and cerebral hæmorrhage were 77% and 29% respectively. These findings seem to coincide well with the general experience of clinicians. They leave us no closer to the powers of prophecy which are often expected by the public, but that may not be altogether a hardship.

[1] *The American Journal of the Medical Sciences*, June, 1948.

THE CONGO RED TEST FOR AMYLOIDOSIS.

AMYLOID DISEASE was once of great interest to clinicians in the days when chronic sepsis was not uncommon and cachectic states were to be seen in every hospital. The many advances that have followed Lister and the rise of bacteriology abolished most of this lingering type of illness, and amyloid disease became one of the almost forgotten conditions. True, students of histology were supposed to be familiar with the characteristic appearance of the tissues, but the diagnostic staining reactions with methyl violet were usually applied only to material obtained at autopsy. Later the subject was reinfused with some life, partly owing to the greater interest taken in renal disease, and partly to the introduction by Bennhold of a test easily carried out on the living patient. However, not so much confidence is felt today in hard and fast classifications of renal disease, and renal amyloidosis is probably not often considered. Bennhold's test depended on the affinity of amyloid tissue for the harmless dye Congo red, which is also remembered as a therapeutic agent for the control of hæmoptysis. The difficulty in this test, as in others, lay in the interpretation of its results, for it was not reliable unless almost the whole of the injected dye was absorbed, and even then unexplained "false positives" occurred. The subject has been brought up again in a paper by Irving J. Selikoff.[1] The author points out that amyloidosis, being a generalized disease, is really of importance, especially in tuberculosis, and the diagnosis cannot always be simply made by clinical means, such as the finding of splenomegaly and hepatomegaly and alteration of kidney function. The great interest taken now in the therapeutic assault on pulmonary tuberculosis, both by local measures and by the use of new drugs, demands that an accurate knowledge be gained of each patient as a whole when his chances of recovery are assessed. One series quoted by Selikoff showed an incidence of 22% in 2000 patients coming to autopsy, and other figures agree with this estimate. Efforts to establish the diagnosis during life have not been much furthered by Bennhold's test, as technical refinements could not dispose of the tendency of the test to miss lower grades of amyloid disease, and to suggest its presence when it was not a feature of the patient's clinical condition. Recent workers, however, considered that 90% or 100% absorption could be regarded as trustworthy evidence of the existence of amyloid disease. Errors were infrequent, though they still occurred, but Selikoff undertook this study in order to find out if more accurate criteria could be established. Sixty-one patients in a large hospital for tuberculosis were studied. All of them had proven osseous or pulmonary tuberculosis, and the Congo red test had shown an absorption of 90% to 100%, when an accepted modification of the original method was used. Clinical evidence was carefully assembled, and biopsy was made of the liver and spleen of a few patients, and of the gingivæ of many others. It has been found that the gums are a frequent, though not invariable, site for amyloid deposits. In general good correlation was found between the results of the test and other evidence, but where conflict was discovered Selikoff found that a simple and useful method was to repeat the Congo red absorption test. When this was done a lower degree of absorption was sometimes found, which in itself cast doubt on the former reading. Absorption of 100% of the dye was found in 33 cases to be quite reliable for all practical purposes. All patients who showed complete or nearly complete absorption invariably gave the same result on later testing. There is in fact a general moral in this, for repetition of an equivocal test of any kind is often found most helpful. This work shows that the test introduced a generation ago is still of value.

[1] *The American Journal of the Medical Sciences*, June, 1947.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Antibiotics and Corynebacterium Diphtheriæ Infections.

L. F. HEWITT (*The British Journal of Experimental Pathology*, August, 1948) tested the protection afforded by streptomycin, penicillin and licheniformin against *Corynebacterium diphtheriæ* infections. Three strains of the *gravis* organism type I were used, and their responses to the antibiotics *in vitro* were first ascertained. Guinea-pigs were infected by either the subcutaneous or intradermal routes, and the drugs were all injected subcutaneously. The untreated control animals died in twenty-six hours; those treated with streptomycin survived more than ten days; those treated with penicillin survived up to forty-seven hours; those treated with licheniformin survived up to fifty-one hours; those treated with diphtheria antitoxin survived more than ten days. Subcultures of the organisms in media containing dilutions of penicillin showed a slow increase in resistance up to 0·5 unit per millilitre. In a similar experiment with streptomycin, however, resistance rose more rapidly and was maintained, and when such strains were used to infect animals the resistance was also exhibited in the response to treatment, the animal dying in twenty-eight hours in spite of a dose of streptomycin which would protect an animal infected with a sensitive strain. The author points out the significance of this result and emphasizes the fact that in its clinical use streptomycin should be given early in effective doses in order to avoid the development of these resistant strains.

Tuberculosis of Rabbits Induced by Droplet Nuclei Infection.

H. L. RATCLIFFE AND W. F. WELLS (*The Journal of Experimental Medicine*, June, 1948) in the first of two studies observed the initial response to infection with varying numbers of tubercle bacilli introduced into the lungs of rabbits on droplet nuclei. A careful technique was developed by which an aerosol flask, to which had been added a single cell culture of *Mycobacterium tuberculosis bovis*, was connected to a cage into which the test rabbits' heads had been admitted, and the infected air was drawn out through an incinerating chimney in order to sterilize it. When an experiment was stopped the system was exposed to ultra-violet light in order to deal with any remaining infected particles before the animals were removed to their individual cages. Groups of sixteen animals were used, and sacrificed at varying times after infection. Lesions appeared after twelve days and were uniform in appearance from the different dosages up to a period of five weeks, but after this period of time differences in progress of the lesions became apparent and seemed to be related to the number of tubercles and the number of bacilli present. It was concluded that the animals varied widely in their capacity to acquire resistance. In the second study these authors report the response of rabbits to reinfection. The animals were infected by small numbers of tubercle bacilli as in the first study, and then were reinfected by exposure to approximately 20,000 bacilli, the time of reinfection varying from two to eleven weeks after the first exposure. Normal controls receiving the large dose of bacilli died after four weeks, and some of the reinfected rabbits succumbed in a similar time—these were animals in whom the interval was short. When the interval before reinfection was five weeks or more, the results suggested that an immunity had developed which apparently prevented reinfection, and this seemed to be produced by inhibition of multiplication of the newly introduced organisms. The development of this immunity did not appear to bear a direct relationship to time, but to progress in steps, and the response to a small initial dose of bacilli in some instances was sufficient to prevent reinfection with the large dose.

Poliomyelitis in the Cynomolgus Monkey.

H. K. FABER, ROSALIE J. SILVERBERG AND LUTHER DONG (*The Journal of Experimental Medicine*, July, 1948) continue their studies on poliomyelitis in the cynomolgus monkey with further observations on exposures confined to the stomach and intestines, with notes on the fæcal excretion of virus. Their technique was designed to avoid contact of the virus inoculum with the upper part of the œsophagus and pharynx, and the material was administered by capsule. In eighteen animals only one infection was established. Virus was recovered from the stools of these animals for a short time after the feeding of the material. Later intracerebral inoculation with virus showed that the animals had not developed any immunity to poliomyelitis. The authors believe that the gastro-intestinal mucosa is relatively impermeable to virus, and that recovery of virus from fæcal material is the result of its excretion into the alimentary tract. The presence of virus in the intestine without infection does not produce immunity to intracerebral inoculation.

Inhibitory Substance to Hyaluronidase.

GEORGE J. FRIOU AND HERBERT A. WENNER (*The Journal of Infectious Diseases*, March-April, 1947) have devised experiments to test the occurrence in human serum of an inhibitory substance to hyaluronidase produced by a strain of hæmolytic streptococcus. They employed the fact that a protein-hyaluronic acid complex will form a typical mucin clot on the addition of acetic acid; the addition of a sample of enzyme hyaluronidase will prevent the production of this clot, and the enzyme can be titrated by this method. Since bacteria produce antigenically distinct hyaluronidases, the neutralization of a known enzyme by a serum may be used to indicate the bacterial origin of the antibody in that serum. Hyaluronidase was prepared from cultures in titres up to 1 in 500. Serum was used diluted in 1 in 10 with distilled water, and the potassium salt of hyaluronic acid was prepared from umbilical cords; the test was carried out with the agents immersed in ice-cold water. The results obtained when the enzyme was elaborated by a hæmolytic streptococcus of group A type 4 showed that the serum of many humans contained an inhibitory substance for this enzyme, and high ratios could be obtained in the case of serum from patients with active rheumatic fever, though such also were obtained in the case of normal serum, and there appeared to be a correlation between the antienzyme and the age of the serum. The inhibitory substance appeared to be specific for the enzyme of this particular streptococcus.

Virulence and Toxigenicity of Serological Types of Corynebacterium Diphtheriæ.

L. F. HEWITT (*The British Journal of Experimental Pathology*, April, 1948) has extended his studies on diphtheria bacilli by comparing the virulence and toxigenicity of the serological types. He used the lethal effect on guinea-pigs as a test of virulence and the skin necrotizing effect as a measure of the toxicity of culture filtrates. Of 208 strains tested, 170 were virulent to the guinea-pig and produced toxin; the 38 avirulent strains were all non-toxigenic. The titre of toxin in culture filtrates reached its highest levels in the *mitis* strains, and the average was lowest in the *gravis* strains. All toxins are neutralized by the same antitoxin. The serological classification of types shows correlation between type and toxigenicity, but some *mitis* types have avirulent subgroups. Some virulent toxigenic *mitis* strains were found to ferment saccharose and the author points out the problem in identification which this creates.

Training or Mutation of Bacteria.

PAUL FILDES AND K. WHITAKER (*The British Journal of Experimental Pathology*, June, 1948) have examined the problem of the reason for the "training" of bacteria to dispense with otherwise essential foodstuffs and whether this might not be due to selection of mutants which were present in the culture before the so-called training was undertaken. The test organism was *Bacterium typhosum*, and the circumstance utilized was its use of tryptophan as a metabolite. It was established that individuals capable of growing without tryptophan were present in normal cultures which require it, and their appearance was not related to the amount of tryptophan in the culture medium. The authors state that this is evidence in support of the suggestion that these are genetic mutants and that methods of so-called training merely develop conditions more favourable to the mutant than to the parent culture.

HYGIENE.

Lung Cancer in Chromate Workers in the United States.

WILLARD MACHLE AND FREDERICK GREGORIUS (*Public Health Reports*, August 27, 1948) present the results of a study, initiated by the health committee of the chromate industry, of the occurrence of cancer of the respiratory system in workers in that industry. The committee represented the five companies in the United States engaged in the production of chromates. The death rate was found to be high. The authors present the following conclusions. Of all deaths in the chromate industry, 21·8% were reported to be

due to cancer of the respiratory system. This ratio was 16 times the expected ratio of 1·8%. The ratios in five of the six plants ranged from 13 to 31 times the normal figure. The crude death rate for cancer of the lung was 25 times the normal figure, the range of excess for the various plants being from 18 to 50 times as great. The high rate for cancer of the respiratory system results in a high rate in the chromate industry for "cancer—all forms". As the number of deaths is small, there are no convincingly significant or consistent abnormalities in the rates for cancer in sites other than the lung. From the available data, it seems that the problem in the chromate industry is limited to cancers of the respiratory system. Experience also suggested that the monochromates might be the compounds responsible for lung cancer. The occurrence of nasal irritation and perforation does not necessarily imply exposure to kinds and quantities of chromium compounds capable of producing lung cancer.

Chronic Carbon Dioxide Poisoning.

H. MEESSEN (*Archives of Pathology*, January, 1948) points out that employees of wine cellars and cold storage plants using dry ice and crews of submarines may undergo prolonged exposure to an atmosphere containing sublethal concentrations of carbon dioxide. He states that, although the clinical, physiological and chemical effects of such exposures have been studied in animals, no data are available on the anatomical reactions elicited in the internal organs. He presents the results of experiments in which dogs, rabbits, guinea-pigs and rats were exposed repeatedly and over long periods to sublethal doses of atmospheric carbon dioxide; post-mortem degenerative changes were found in the lungs, the liver, the kidney and the brain. He emphasizes that these lesions were the result of the increased carbon dioxide tension and were not caused by anoxæmia. The results suggest that it is not impossible for similar changes to develop in man under special conditions of exposure.

Diphtheria Outbreaks due to Convalescent Carriers.

C. E. DOLMAN, VIVIENNE G. HUDSON AND DONNA E. KERR (*Canadian Journal of Public Health*, August, 1948) describe three outbreaks of diphtheria in British Columbia, two of small dimensions, in which the source of infection appeared to be a carrier who was known to have recovered from an attack of diphtheria several weeks previously. In the larger outbreak eleven typical cases of diphtheria were identified, two of them fatal, with two mild cases and eighteen carriers. All the subjects involved were contacts, direct or indirect, of a young boy who had been released from quarantine after a severe attack of diphtheria, on the basis of negative findings in nose and throat swabs examined on three successive days. The authors point out that it is now recognized that artificial diphtheria immunization will not guarantee full protection to any subject even for a few years, still less for a lifetime. *Corynebacterium diphtheriæ* has undiminished versatility in morphological characteristics, colonial form, serological type and toxigenic capacity. Further, the development of improved culture media has not lowered earlier estimates of the carrier rate. Public health regulations customarily lag well behind authenticated laboratory findings. In view of their findings the authors recommend that convalescents should not be discharged from hospital until a series of five examinations of nose and throat swabs on alternate days has given negative results. After the provisional withdrawal of quarantine restrictions, follow-up examinations of swabs might with advantage be made at intervals of one week for a short time before the patient is finally released from observation.

Tuberculosis Mortality in the United States.

E. H. HALPIN (*Public Health Reports*, August 6, 1948) presents the data for 1946 on tuberculosis mortality in the United States and in individual States as well as corresponding data for 1945 and for the period 1942-1944. In 1946 the death rate for tuberculosis (all forms) in the United States was 36·4 per 100,000 of population, as compared with the 1945 rate of 40·1, an apparent continuance of a downward trend. In 1946, 50,911 deaths from tuberculosis were registered. As in other years, the death rates for white persons were considerably lower than those for the non-white group, the difference amounting to 63% for males and 74% for females. The tuberculosis mortality rate for males was 72% higher than that for females. Amongst males, the death rates rose steadily from their lowest values in early childhood to their highest values at the ages of sixty-five to seventy-four years. Amongst females, the rates rose during the adolescent and young adult ages, but showed a decline in the middle adult ages; the rates for non-white females, however, did not show the same rise in the older adult years as appeared in the rates for white females. Between 1945 and 1946, decreases were recorded for almost every age group for each of the four race-sex groups. The largest of these decreases occurred in the rates for males aged fifteen to thirty-four years, although this may be partially attributed to a change in the size of the male population resulting from the return of overseas armed forces. Respiratory tuberculosis accounted for approximately 92% of all deaths attributed to tuberculosis.

Rheumatic Heart Disease.

R. W. QUINN, J. H. WATKINS AND J. P. QUINN (*American Journal of Public Health and The Nation's Health*, August, 1948) report the results of a survey of rheumatic heart disease amongst a sample of rural, semi-rural and urban school children in Connecticut. The rheumatic heart disease incidence rate for the sample of rural children examined was 1·6%, for semi-rural children 1·4%, and for a sample of urban children 2·45%. These differences are stated to be not statistically significant. The incidence rate for children in one city was significantly higher than the rural rate or the combined rate of the other urban centres studied. The reasons for these differences are not known, but they accord with the idea that this disease flourishes more in certain localized places than in others. The incidence rate for urban children living in low rental areas was 2·5%, in medium rental areas 2·7%, and in high rental areas 1·4%. These differences are not statistically significant and, furthermore, there was no significant difference between the incidence rate among urban children in a low rental area and the similar rate for rural children. One notable feature was the effect of crowding within the home which was apparently an important factor influencing the prevalence of the disease; the rate in crowded homes was almost twice that in non-crowded homes. The other findings of the survey failed to show significant differences, with the exception of a familial history of rheumatic fever or rheumatic heart disease which was significantly higher among children with rheumatic disease.

Economic Status and Tuberculosis Mortality.

M. TERRIS (*American Journal of Public Health and The Nation's Health*, August, 1948) states that numerous studies have shown the close correlation of occupational class and economic status with tuberculosis mortality and stability. Analysis of tuberculosis mortality by age, sex and economic status in the city of Buffalo during the years 1939-1941 indicates that the inverse correlation of economic status with tuberculosis mortality is greater for adult males than for adult females. It is concluded therefore that economic status *per se* is not the only factor involved, but that conditions of occupation probably have an important effect. The author discusses various aspects of occupational environment, and suggests that the factor of physical overstrain plays a significant role. He recommends that similar studies be conducted in other cities to test the general validity of the findings, and that further studies be instituted to isolate the various factors, if possible, in order to determine their relative influence on tuberculosis mortality.

Margarine and the Growth of Children.

H. LEICHENGER, G. EISENBERG AND A. J. CARLSON (*The Journal of the American Medical Association*, February 7, 1948) state that growing children experience normal growth in height and weight when their diets contain only fortified margarine as table fat, as shown by comparison with children fed on similar diets with butter as the source of table fat and by comparison with standard height and weight tables. Whether the greater part of the fat of the diet is derived from vegetable or animal sources has no effect on growth and health as shown by changes in height and weight and health records of children observed over a two-year period. The health of 267 children was uniformly good during a two-year period so far as serious illness was concerned, regardless of whether margarine or butter was the source of the greater part of the fat in the diet. The authors consider that, if there is a growth factor present in butter which is not present in margarine, there is no evidence in the present study that such a factor plays any important part in the growth of children as determined by increases in height and weight. Their two-year study indicates that margarine is a good source of table fat for growing children. They found that children readily accepted margarine as a table spread when it was coloured and served in pats.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on August 4, 1948, at the Medical Society Hall, East Melbourne, MAJOR-GENERAL F. KINGSLEY NORRIS, C.B.E., D.S.O., the President, in the chair.

Medico-Legal Problems.

DR. K. M. BOWDEN, lecturer in forensic medicine at the University of Melbourne, read a paper entitled "Medico-Legal Problems Based on Experience at the Morgue" (see page 12).

PROFESSOR P. MACCALLUM opened the discussion by expressing thanks to Dr. Bowden. He emphasized the importance of Dr. Bowden's admirable investigation of the significance of blood alcohol levels. He also directed attention to what Dr. Bowden had said about the factor of respiratory obstruction in deaths under anæsthesia. Professor MacCallum also said that those present should be glad that Dr. Bowden had taken up the work at the morgue, because it was of the utmost importance to the medical profession and to the public that medico-legal work should be in the hands of people who knew their business thoroughly.

DR. GEOFFREY KAYE said that Dr. Bowden had almost qualified to be an honorary member of the Society of Anæsthetists, and he thanked him on behalf of the anæsthetists for what he had said. Dr. Kaye went on to say that the anæsthetic death risk was a small one, approximating one death for 1200 general anæsthetics. At a large general hospital in Melbourne, over a ten-year period, the rate was one in 1270. Potent weapons had to be used carefully, and in order that patients might have the advantages of modern advances in anæsthesia, the anæsthetists must know what they were doing. Dr. Kaye was of the opinion that young medical graduates should have the principles of anæsthesia instilled into them when they were doing their practical work as resident medical officers. He agreed with Dr. Bowden that the asphyxial deaths must be stopped, and that artificial aspiration must not be carried out when the breathing passages were not clear of foreign material. He recommended that emergency surgery should be put off as long as possible after a meal, to avoid, so far as was possible, the risk of regurgitation.

DR. IAN WOOD also congratulated Dr. Bowden. He said that it was helpful to have Dr. Bowden's assurance that he was sympathetic to doctors who found themselves involved in deaths that came under the notice of the coroner. It should be remembered that younger members of the profession had to accept great responsibility and that they felt that responsibility; it was, therefore, reassuring to know that Dr. Bowden could advise the coroner of the doctors' difficulties. Treatment had to be undertaken at times which involved great risk, in order to give the patient the only possible chance of survival. Dr. W. G. D. Upjohn and many others, for a long while past, had drawn attention on many occasions to the anæsthetic risk when food was present in the stomach. Dr. Wood then commented that Dr. Bowden had skimmed over his reference to the work in connexion with the significance of the blood alcohol level; Dr. Wood was well aware of the immense amount of work that had been necessary to reach the conclusions made by Dr. Bowden.

DR. LEONARD BALL, speaking on behalf of the surgeons, said that they were glad to know that the coroner assessed the risk from the surgeons' point of view and protected honest judgement, even if after the event that judgement could be impugned.

DR. G. RALEIGH WEIGALL spoke of the risk during the recovery period after an operation, and asked Dr. Bowden to define the period within which a death could be regarded as occurring "under an anæsthetic". Dr. Weigall thought that the attention of medical practitioners should be drawn to the way in which, in short-staffed hospitals, patients were left unattended before they were able to look after themselves. Naturally they were not unattended until they were regarded as being out of the anæsthetic; but if a patient was left alone before completion of recovery accidents could occur, and it was hard that the anæsthetist should be held blameworthy. Finally, Dr. Weigall asked Dr. Bowden whether he could supply a scale of blood alcohol levels indicative of ability to drive a motor-car and to carry out other activities.

DR. ERIC GANDEVIA said that the asphyxial deaths often involved young patients, children or babies, and were often the outcome of relatively minor surgical or dental procedures. Dr. Bowden had not said where the tragedies had occurred—for example, in doctors' consulting rooms or in the homes of the patients. Dr. Gandevia was of the opinion that, wherever an anæsthetic was given, facilities should be available to cope with emergencies, as they would be in a properly equipped operating theatre. In conclusion, he affirmed that there were no "minor anæsthetics", even if it were conceded that there were minor operations.

DR. R. S. HOOPER also thanked Dr. Bowden for his paper, and said that he was glad to know that the coroner's court in Victoria was fulfilling its full duty as a court of justice. Dr. Hooper thought that the management of the unconscious patient was not always well handled, and should be the subject of greater attention in the medical curriculum.

DR. J. COHEN asked Dr. Bowden to supply information as to what proportions of anæsthetic deaths had occurred on the operating table, in the ward, in private hospitals and in public hospitals.

DR. C. H. DICKSON contributed some comments on the medico-legal aspects of deaths in *Workers' Compensation Act* proceedings.

Major-General Norris, from the chair, thanked Dr. Bowden for what he regarded as an outstandingly valuable paper. It was apparent that, fostered and guided by people such as Dr. Bowden, the kindness of the coroner was exerted to protect members of the medical profession from unmerited blame. Major-General Norris too was of the opinion that Dr. Bowden's work on the significance of alcohol levels in the peripheral blood was a valuable contribution. He went on to say that he was not at all satisfied that so much attention should be paid to didactic instruction of young graduates in anæsthesia; all they should know was how to give ordinary anæsthetics; ultra-modern anæsthetics should be reserved for the use of ultra-modern anæsthetists. At present, seven lectures out of a total of about forty in medicine were devoted to the theory of anæsthesia. Dr. Bowden had shown that far too frequently doctors had failed in the much more important responsibility of caring adequately for the welfare of their patients.

Dr. Bowden, in reply to Dr. Gandevia and Dr. Cohen, said that nearly all the reports of deaths under anæsthesia came from hospitals, and only an occasional one or two from dental surgeries or private homes. According to the carefully worded statements sent in for the information of the coroner by the doctors concerned, it was apparent to Dr. Bowden that it was seldom recognized that patients had respiratory obstruction, and that the treatment was often misdirected to overcome cardiac failure instead of attention's being paid to the state of the respiratory passages. In reply to Dr. Weigall, Dr. Bowden said that it was impossible to be dogmatic about the significance of the blood alcohol levels, as tolerance for alcohol and idiosyncrasy to alcohol were difficult to evaluate. Speaking broadly, he was of the opinion that 100 milligrammes of volatile alcohol *per centum* in the peripheral blood corresponded with the average cocktail level of vivacity and talkativeness; that at 200 milligrammes *per centum* the ordinary layman would recognize that the subject was "drunk"; that at the 400 milligramme *per centum* level most people would be comatose; and at above 400 milligrammes *per centum* one would expect death from acute alcoholic intoxication. Dr. Bowden added that the defence put up by solicitors seemed to him to be ridiculous at times; motor-car drivers were taken to the doctor's surgery, and it was only occasionally that blood specimens were taken and their alcohol content estimated. In the absence of that scientific evidence, defences such as the effects of minor head injuries and the carbon monoxide content of the air in the motor-car had succeeded when the evidence was strongly suggestive of drunkenness as the real cause of the accident. Unfortunately at present it was not legal to take a blood specimen without the consent of the patient. It was sufficient to obtain two millilitres of blood and cork it securely in a test tube; the specimen could be kept overnight in a refrigerator and next day it could be analysed. Dr. Bowden then expressed his gratitude to Dr. Ian Wood and the assistants at the Walter and Eliza Hall Institute, who had devoted a great deal of time and attention to helping him with his work in connexion with the blood alcohol levels. Dr. Bowden said that a number of the deaths had occurred as long as an hour or more after the operation was completed and the patient had been back in his bed. In his opinion these deaths should not happen. He emphasized the point that all classes of doctors and all classes of hospitals were represented in the material that he had analysed. In conclusion, he pointed out that it was frequently desirable to conduct an inquest, even though he and the coroner were satisfied as to the cause of death, because the interested parties were perfectly entitled to have a public inquiry to decide just how the death was explained.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held on May 7, 1948, at the Institute of Medical and Veterinary Science, Adelaide.

The Pleuropneumonia Group of Organisms and their Relationship to Arthritis.

MRS. K. W. DRAPER said that the pleuropneumonia group of organisms occupied a position between the viruses and the bacteria. They passed through bacterial filters, but would grow on special bacteriological media. The strain causing bovine pleuropneumonia had been most studied in the past, being fairly easy to cultivate. The rat, mouse and human strains needed more complex media for their growth. The organisms were viewed with dark-ground illumination, as they did not stain with ordinary stains. Most diseases due to pleuropneumonia organisms affected the joints, and it therefore seemed likely that the group might play some part in human arthritis. A mouse strain had been isolated in America which caused arthritis in 100% of mice injected intravenously. Although the organisms had not so far been isolated from joints of humans, they could often be cultivated from the female genital tract, especially when pelvic disease or arthritis was present. It also seemed probable that they might have some connexion with non-specific urethritis, and even more probable that they were the causative agents of Reiter's syndrome.

Neurosyphilis.

A symposium on neurosyphilis was given by three members of the Club.

The Treatment of Neurosyphilis with Rat-Bite Fever.

DR. ORDE POYNTON, in opening the symposium, gave a brief history of syphilis and *dementia paralytica*. Evidence suggested that syphilis was introduced into Europe from the West Indies in 1492; that syphilis, pinta and yaws were closely related; and that it was possible that the original host of the treponema was the blame of South America. Those were important arguments in favour of the treponema's having a life cycle involving an intracellular phase which had not yet been elucidated. The essentially syphilitic nature of *dementia paralytica* was a relatively recent discovery; the syndrome was not distinguished from other dementias until 1822, and its connexion with syphilis was not suggested until about 1900, while proof had to wait until 1913, when Noguchi identified treponemata in the brain substance of deceased subjects. Actually *dementia paralytica* was one of the most syphilitic of syphilitic lesions; in it parenchymatous destruction was accompanied by a chronic meningitis and vascular reaction. About 3% of untreated patients with acquired syphilis developed meningo-encephalitis, and it was also a feature of late congenital syphilis.

Dr. Poynton then gave a brief account of the pathological findings in untreated neurosyphilis, correlating them with the symptoms and laboratory findings. He went on to summarize the history of treatment, emphasizing the failure of arsenicals, which had proved valuable in primary and secondary syphilis. As early as 1887 Wagner von Tauregg had suggested malarial therapy for *dementia paralytica*, the suggestion being based on the observed fact of substantial remissions following an acute febrile illness; but it was not until 1917 that he put his suggested therapy into practice, obtaining six remissions in nine cases. Later, in 400 cases he reported complete remissions in one-third. Tuberculin, streptococcal vaccine, typhoid vaccine and other protein substances had been used, but none had proved so effective as malarial infection, the only rival to which was the fever produced in one of the several designs of cabinet available.

Dr. Poyton then described an experiment in which infection with *Spirillum minus*, the causative organism of rat-bite fever, was used to treat forty patients with *dementia paralytica* in Malaya, where the condition was common, especially among the Chinese. Malarial therapy was valueless, as nearly all subjects were immune from previous infection, and its use in areas where transmitting mosquitoes were prevalent created special problems. Rat-bite fever was common in Japan, but sporadic cases were encountered throughout the Far East. As the ætiology of the disease had been called into question by American workers, and as infection had been found to give titres in the Weil-Felix reaction, work on those problems was combined with the treatment. A particularly virulent strain of *Spirillum minus* from a human subject was maintained by passage in guinea-pigs, and the patients were infected by the intravenous injection of dilutions of guinea-pig blood, as it was found that scarification sometimes caused extensive ulceration. All subjects were successfully infected, and in a high proportion satisfactory fever was produced. In most cases the fever was terminated with "Novarsenobillon" after two relapses, as a result of which the patient had about six days with fever reaching 104° F. and three days with fever reaching 102° F. At the height of the fever some patients showed considerable collapse, but no immediate mortality was recorded. The only serious complication encountered was nephritis, from which four patients died in uræmia. The results could not be compared with those obtained in western countries, as in nearly all cases the condition was advanced when the subjects were first examined, their physical condition was usually poor on account of malnutrition, and most had received no anti-syphilitic therapy at the time of infection. However, six patients showed a remission comparable to that obtained with malaria, and another small group showed some improvement, while the remainder gained nothing from the treatment. That *Spirillum minus* was responsible for rat-bite fever in Malaya had been fully established. Elaborate clinical records of all aspects of these infections were kept, together with material from the fatal cases, for study of the pathology of the disease; but they had been lost during the Japanese occupation of Malaya.

Penicillin and other Therapeutic Agents in Neurosyphilis.

DR. IAN MARTIN, discussing penicillin and other therapeutic agents in neurosyphilis, enunciated several general principles of treatment. (i) Destroyed neurons could never be replaced, though less severely damaged neurons might recover, and further damage might result from fibrosis and endarteritis. (ii) Structures and functions of the brain most recently acquired phylogenetically were the most susceptible. (iii) The same structures and functions recovered last and least. (iv) When there was no specific cure of a disease, a combination of the most potent agents was required. (v) When no such combination would easily produce cure in a high percentage of cases, the campaign must be long and intensive. Therefore early diagnosis and prompt, energetic and sustained treatment were especially desirable in neurosyphilis.

Experimental work was then cited to provide a rational basis for therapy. (i) Hyperpyrexia had been shown to increase the efficacy of penicillin in a test-tube experiment with a standard culture and dose in experimental syphilis in rabbits. (ii) Patients with early syphilis, cured by penicillin, had returned with relapse or reinfection, in some instances clearly the latter; consequently an experiment was carried out on rabbits which dramatically demonstrated the efficacy of penicillin against syphilis. (iii) The total curative dose in units per kilogram had been determined for the Romansky formula in experimental syphilis in rabbits. Because of the small dose required, the original material had to be diluted, its efficacy thus being lessened. Yet the C.D. 50 was 1400 units per kilogram (without fever, C.8 1); thus the superiority of that preparation was demonstrated.

Dr. Martin then referred to clinical reports, chief among which was the latest cumulative report, from Stokes *et alii*, upon the use of penicillin alone. The main points for this were the following. (i) The best effect seemed to be gained in up to 120 to 200 days after the course, when repeated courses, if necessary, were best given. (ii) There was little tendency to relapse (11%). (iii) Clinical improvement was judged with malaria and alone, and the effect on the cerebro-spinal fluid abnormalities was always greater with malaria. (iv) Great symptomatic improvement was obtained in 46% of cases of general paralysis of the insane, in 35% of cases of tabo-paresis, in 33% of cases of tabes and in 47% of cases of meningo-vascular syphilis. The cerebro-spinal fluid findings returned to normal or nearly normal in 33%, 40%, 55% and 55% of cases respectively. Penicillin combined with malaria was more effective than penicillin alone. Untreated, 90% of patients died within five years of diagnosis.

Discussing the combined course of therapy, Dr. Martin said that the administration of Lugol's iodine and lipo-soluble bismuth was commenced. The onset of malarial rigors was timed to coincide with the sixth bismuth injection (injections were given at intervals of five days). Thiobismol was used to produce a tertian fever. On the first day of the rigors, the first of fourteen daily injections was given of 300,000 units of penicillin (Romansky), and tryparsamide, one gramme, two grammes, then three grammes, every five days for seventeen injections (a total of 48 grammes). If the patient was not fit for malarial therapy it was omitted. After a rest of one month the course was repeated; but malaria was used only once.

Dr. Martin finally indicated the contraindications and dangers of such treatment, placing the main emphasis on the optic nerve Herxheimer reaction, which could be minimized only by a rigid routine of inquiry about subjective visual symptoms and by the performance of the "confrontation test" before each injection of penicillin and tryparsamide. Administration of the drug was stopped immediately, to be resumed later if necessary after further bismuth therapy. Dr. Martin also referred to the use of BAL at the onset. He said that the unsatisfactory results so far obtained with less heroic treatment had urged him to explore the possibilities of such a comprehensive campaign.

Serology.

MR. R. A. W. SHEPPARD discussed the peculiarities of the serological tests for syphilis. He said that the tests were rather unique, in that the antigens used were not suspensions or extracts of the infecting agent, the *Treponema pallidum*, but cholesterinized alcoholic extracts of normal heart muscle. That meant that in the strict sense of the word the reactions could scarcely be classed as "specific". It was only because in the early days the results were confirmed by clinical findings that the tests had come into being. The sensitivity of the tests had been increasing over the years, and it had been found that a limit must be placed on the sensitivity, or else a state would be reached in which all specimens of serum would give a positive result. Even the most reliable tests available gave occasional "false positive" results. That was to be expected in syphilis-like diseases, and had come to be expected fairly frequently in diseases such as leprosy, filariasis and malaria, and less frequently in many other pathological states. What was not generally realized was that serum from a small percentage of apparently normal persons might yield a positive reaction. The reason was as yet unknown; the result had been attributed to "lability" of the serum proteins. That rendered highly dangerous the assumption that, since persons who had syphilis gave positive reactions to serological tests, those who gave positive reactions to serological tests had syphilis. Only after intensive, thorough examination of the patient, with attention to history, physical and radiological studies, cerebro-spinal fluid examination and repeated serological tests, was the physician qualified to make his diagnosis. Then the serological tests must take their proper place as one factor in the chain of evidence.

A MEETING of the Medical Sciences Club of South Australia was held on June 4, 1948, at the Institute of Medical and Veterinary Science, Adelaide.

The Statistical Analysis of Biological and Medical Data.

MISS H. M. FERRES said that statistics were dependent on a study of populations and their variation. An example was given of a population of heights, which could be represented by a normal curve, the form of most biological data. That curve was completely specified by the arithmetic mean and the standard deviation, which measured the scatter of the individual values about the mean—in other words, the variability of the data. The probability of a value of the population's exceeding the mean by $\pm$ 1·0 × standard deviation was one in 3·3 times; by $\pm$ 2·0 × standard deviation, one in 20 times ($P = 0{\cdot}05$); by $\pm$ 2·6 × standard deviation, one in 100 times ($P = 0{\cdot}01$). The 5% and 1% probabilities were taken as low and high levels of significance respectively. An example was given with two treatment means differing by 50%. Its significance was tested by the *t*-test; it was found to be not significant, owing to the high variability within the samples.

Miss Ferres went on to say that the standard deviation of a mean, $\sqrt{\frac{\Sigma(x-\bar{x})^2}{n(n-1)}}$, could be decreased by increased replication or by decreasing the variation. To illustrate the latter, the previous example was taken again, and it was shown that, by an improved design, the treatments could have been shown to be significantly different. Care had to be taken to ensure random examples which were representatives of the whole; otherwise the conclusions applied only to the selected groups chosen. The analysis of variance divided the total variance into parts contributed by the various treatments and the remainder, which was the estimate of experimental error, and was used to calculate the significance of treatment. The factorial design was a modification of the analysis of variance by which more than one factor was varied at a time. That design had the advantage of internal replication and measured any interactions. It was of particular importance at the beginning of an investigation.

Some uses of the χ^2 test were mentioned: (i) to test the goodness of fit of frequency data with a known hypothesis—for example, segregation ratios in genetics; (ii) to test the goodness of fit of some data to a known distribution; (iii) to test for the existence of association. The correlation coefficient *r* measured the degree of association between two variables; *r* ranged from +1·0 to −1·0, and its significance could be determined from tables. Where correlation existed between two variables, one could be expressed in terms of the other by means of a regression equation.

Liver Function Tests.

MISS M. FREEMAN said that a group of biochemical tests was necessary to distinguish between hepato-cellular jaundice and that due to external obstruction. The cephalin flocculation test, the alkaline phosphatase level and the ratio of serum albumin and globulin gave different pictures for the various clinical conditions. Serum from patients with hepatitis gave a strongly positive response to the cephalin flocculation test and had a moderately raised alkaline phosphatase level. Serum from patients with chronic hepatitis had an inverted albumin : globulin ratio, whereas in acute hepatitis the ratio was normal. In obstructive jaundice the cephalin flocculation test produced a negative response, the ratio of serum proteins was normal, and the alkaline phosphatase level was higher than in cases of hepatitis. The quantitative Van den Bergh test was useful for following the course of the jaundice and for the detection of latent jaundice, but would not distinguish between the types. The hippuric acid excretion test was a delicate index of liver dysfunction, but gave no information concerning the cause. Amino acids might be detected by chromatography in urine from patients with liver failure.

Correspondence.

A CORRECTION.

SIR: The current number of the journal contains an account of the clinical meeting of the British Medical Association, New South Wales Branch, recently held at the Royal North Shore Hospital of Sydney, and among the cases shown was one by myself, namely, mucocele of the left orbit. This account appears on page 702. The conclusion of the paragraph reads: ". . . the mucocele which was closely associated with the sphenoidal sinus . . ." This is an error in the notes supplied by the hospital and should read frontal sinus and not sphenoidal sinus.

Yours, etc.,
ERIC P. BLASHKI.

Hengrove Hall,
193, Macquarie Street,
Sydney.
December 13, 1948.

A NATIONAL MEDICAL SERVICE.

SIR: I am opposed to the introduction of the national medical service as proposed by the Federal Government at the present time for a number of reasons, and would welcome the opportunity to publicize them. They are as follows:

1. Insufficiency of what our present rulers may easily understand, when presented in their language, namely, the medical workshops and tools of our trade. The present lack of adequate and efficient hospital accommodation goes back far beyond the present Government, for many of our past rulers have sinned and come short in their failure to supply such. But at no time in the history of this country have the coffers of the Treasury been so full, and this alone makes the obligations of providing adequate, efficient and up-to-date hospitals, clinics *et cetera* more incumbent on this present Government.

Tuberculosis, arthritis, mental disorders *et cetera* are calling not for free or half-price attention at surgeries or homes, but crying out for places to be nursed back, if possible, to a useful life. These two former diseases claim 20% of our invalid pensions and the weight of invalidity could be progressively lifted by adequate hospitals and clinics. Our aging population, as evidenced by the doubling of old-age pensioners from 1920 to the last census, when the population increase has not been commensurate, reveals

the necessity for increasing medical care to increase the effective working age of an aging population so as to prevent or postpone invalidity. This increase in longevity will increase the demand for hospital accommodation. Every hospital has a large waiting list, in some cases 800 to 1000 patients. In the chronic rheumatic diseases there are not 30 beds put aside in all Australia, whilst in Sweden there are over 3000 beds for these cases alone.

The tragedy of an increasing invalidism due to the chronic diseases could be mitigated by adequate hospitalization and financial drain on the community relieved thereby, but it will be perpetuated by the *National Medical Service Act*.

2. The basic need of the sick community at present is therefore adequate hospital accommodation, the therapeutic tools for effective treatment and the staff for efficient working. The moneys, now for the first time available, will be deviated to the National Medical Service and away from what we as medical men know by our experience and practice is the most pressing need of the hour. Let these fundamental services be established and every sick person needing hospitalization in efficient hospitals be reasonably able, in a short time, to obtain that accommodation, and the stage will be set for a march forward to provide a so-called free service for all with the terms of medical service in conformity with our ideals of the best standards of medical practice. The Government is politically minded and not medically minded, otherwise the present alarming hospital shortage and need of tremendous alterations, besides additions, would not blind their eyes to the most urgent medical problem of today.

3. The increase of secretarial staff needed by doctors will place greater pressure on the already meagre supply of efficient females and may divert a number from the pressing and urgent need of the nursing profession. Even if only 1000 extra females are required by our 6000 doctors, the cost will be in the vicinity of £5000 per week or more, or over £200,000 per year. This must be provided for by increased work by a profession already burdened with excessively long hours to keep up the present standard of income. Further, the deviation of manpower for the administration of the act would further reduce the available manpower pool and therefore the act can be condemned because of this important reason. It does seem stupid for a government to deliberately increase this present manpower vacuum.

4. The scheme cannot add to the general standard of medicine as practised by medical practitioners, but by perpetuating the present lamentable shortage of hospital accommodation with an increasing and aging population will actually reduce the standard, until the practitioners' Golden Age appears when the bed requirements for the sick with facilities for proper treatment will be adequate.

5. Our Federal Council are rightly concerned about the terms of service in such a national medical service, as the failure to incorporate fundamentals essential to medical practice would be a colossal blunder to all parties concerned, but the failure to point out and press for the urgently necessary hospital accommodation as the foundation of any adequate service before final medical implementation may leave the profession standing indicted before the bar of public opinion, as being more concerned about the terms of service than with the termination of the present tragic lack of hospital accommodation for those fettered by severe or prolonged disease.

The general public are very interested in the absence of adequate hospital accommodation and insistence on adequacy, within reason, as the prelude to the operation of a mutually planned scheme, would be favourably received by Press and public. We are our sick brother's keepers, capable by training and practice to direct an untrained government in the medical path it should follow and in the proper sequence of medical priorities and requirements.

In conclusion let me quote from the *British Medical Journal* editorial, March 20, 1948, page 551:

> Both the Government and the medical profession wish to see that the public has an efficient medical service. The Government claims that their Act will provide this, but we repeat once more that if this Act, even amended, comes into operation on July 5 the public will be disillusioned, and will have a less efficient medical service than at present, for the reason that it will be impossible by July 5—or even for years to come—to provide the necessary number of hospitals, doctors, nurses, dentists and other health workers, not to mention health centres. We suggest that it is still not too late for the Government to review this position so that both the public and the medical profession may co-operate in their fight against disease and in the maintenance and promotion of health.

Our answer should be at present: "No cooperation without the workshops and tools of trade."

Yours, etc.,
L. J. A. PARR.

135, Macquarie Street,
Sydney,
December 13, 1948.

AN APPRECIATION.

SIR: I feel sure that members of the New South Wales Branch will be pleased to read the following letter which I have received from Lord Webb-Johnson, President, Royal Medical Benevolent Fund.

1 Balliol House, Manor Fields,
Putney, London, S.W.15,
3rd November, 1948.

The President,
New South Wales Branch,
British Medical Association,
135 Macquarie Street,
Sydney.

My dear President,

I want you and your fellow Members of the New South Wales Branch of the British Medical Association to know that your generous action in sending Food Gift Parcels to the Beneficiaries of the Royal Medical Benevolent Fund is most deeply appreciated by the Committee of the Fund as well as by the recipients.

We are constantly receiving letters telling us how deeply touched the recipients are, not only by the generosity, but also by the kind thought of professional colleagues so far away. It makes you all very near and dear to us.

With cordial and affectionate greetings and warmest thanks.

I remain,
Yours very sincerely,
(Sgd.) WEBB-JOHNSON,
President.

I take this opportunity of thanking those members who have so generously contributed to the "British Medical Association Food Parcels Fund" throughout the last twelve months. Their ready response to Council's appeal has been the means of bringing much happiness to those of our colleagues and their dependants overseas who have been less fortunate than ourselves.

Yours, etc.,
A. M. McINTOSH,
President, British Medical Association, New South Wales Branch.

135, Macquarie Street,
Sydney,
December 8, 1948.

Congresses.

AUSTRALIAN AND NEW ZEALAND ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE.

THE following is the programme for Sections I and N—Medical Science, National Health and Physiology—of the twenty-seventh meeting of the Australian and New Zealand Association for the Advancement of Science to be held at Hobart from January 12 to 19, 1949. The discussion theme will be "The Control of and Resistance to Infection".

Thursday, January 13.—11.15 a.m.: Presidential Address—"The Nature of Immunity", Professor F. M. Burnet.

Friday, January 14.—Bacteriological and physiological aspects of the discussion theme. 9.30 a.m.: "Some Problems of Cross-Infection", Professor Sydney Rubbo. 11 a.m.: "The Relation of Nutrition to Infection", Dr. F. W. Clements. 12 noon: "The Role of Histamine in 'Non-Specific' Immunity", Professor E. R. Trethewie.

Saturday, January 15.—9.30 a.m.: A demonstration of experimental and clinico-pathological material in relation to the theme arranged by Dr. W. R. Pitney, Royal Hobart Hospital, Hobart. A film and talk on plastic surgery in relation to the theme by Dr. B. K. Rank. Clinical notes

and cases by Dr. Campbell Duncan and Dr. G. A. Robbie. Welcome to Dr. Pett.

Monday, January 17.—Clinical aspects of the theme. A. Medical aspects. 10 a.m.: "Pertussis", Dr. Stanley Williams. 11 a.m.: "Prophylactic Use of Rubella γ Globulin", Dr. H. McLorinan. 12 noon: "Some Problems of Tuberculosis", Sir Sidney Sewell. B. Surgical aspects. 2 p.m.: "The Control of Prolonged Sepsis", Dr. R. Officer. 3 p.m.: "Peritonitis", Dr. E. A. Hedberg.

Tuesday, 18th January.—C. Obstetric aspects. 9.30 a.m.: "The Resistance to and Control of Infection in Obstetric Practice", Dr. Arthur Hill and Dr. Hildred Butler, of Melbourne. Experimental research aspects of the theme. 2 p.m.: "Experimental Transmission of Infectious Diseases to Marsupials", Dr. A. Bolliger. 3 p.m.: "Pathology of Certain Protozoa Infections in Marsupials", Dr. T. Clive Backhouse. 4 p.m.: "Experimental Pertussis in the Mouse", Dr. Stephen Fisher.

Wednesday, January 19.—Public health aspects of the theme. 9.30 a.m.: "Modes of Spread of Infection and their Control", Dr. I. M. Mackerras and Dr. M. J. Mackerras. 10.45 a.m.: "Rheumatic Fever in its Public Health Aspects", Dr. A. R. Southwood. 12 noon: "'B.C.G.': A Promoter of Public Health", Dr. J. Tremayne.

Obituary.

REGINALD ARTHUR PARKER.

We regret to announce the death of Dr. Reginald Arthur Parker, which occurred on November 29, 1948, at Melbourne.

ESMOND SHIRLEY JOSKE.

We regret to announce the death of Dr. Esmond Shirley Joske, which occurred on December 4, 1948, at Melbourne.

ALEXANDER KENNETH MacLACHLAN.

We regret to announce the death of Dr. Alexander Kenneth MacLachlan, which occurred on December 8, 1948, at Stockton, New South Wales.

FRANCIS RUPERT BENSON.

We regret to announce the death of Dr. Francis Rupert Benson, which occurred on December 9, 1948, at Brisbane.

Nominations and Elections.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Chazan, John Jacob, M.B., Ch.B., 1946 (Polish School of Medicine, Edinburgh), Royal Melbourne Hospital, Parkville.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

D'Arcy, Arthur Frederick Joseph, L.M.S.S.A., 1927 (London), 17, Gordola Road, North Narrabeen.

Hall, Peter Francis, M.B., B.S., 1947 (Univ. Sydney), 265, Edgecliff Road, Edgecliff.

Harris, Lelia, M.B., B.S., 1948 (Univ. Sydney), Rachel Forster Hospital, Redfern.

Martin, James Matthew, M.B., B.S., 1945 (Univ. Sydney), corner of Robert Street and Eastern Valley Way, Willoughby.

Holland, John Angus Beveridge, M.B., B.S., 1948 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Solomon, Ivor, M.B., B.S., 1936 (Univ. Bombay), 245, Darling Street, Balmain.

Wilkins, Trevor Douglas, M.B., B.S., 1948 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Wonders, Ivor Herbert Richard, M.B., B.S., 1948 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Diary for the Month.

JAN. 5.—Western Australian Branch, B.M.A.: Council Meeting.
JAN. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 11.—New South Wales Branch, B.M.A.: Council Quarterly.
JAN. 13.—South Australian Branch, B.M.A.: Council Meeting.
JAN. 13.—Victorian Branch, B.M.A.: Organization Subcommittee.
JAN. 14.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 17.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
JAN. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 20.—Victorian Branch, B.M.A.: Executive Meeting.
JAN. 26.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 28.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee (with representatives of Special Groups).
FEB. 2.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 2.—Victorian Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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